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THE VETERINARY JOURNAL

A MONTHLY REVIEW OF VETERINARY SCIENCE

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EDITOR:

PROF. SIR FREDERICK HOBDAY, C.M.G., F.R.C.V.S., F.R.S.E.

DR. MED. VET. h.c. (ZURICH),
HONORARY VETERINARY SURGEON TO HIS MAJESTY THE KING,
OFFICIER DU MÉRITE AGRICOLE (FRANCE),
CAVALIERE DEI SS. MAURIZIO E LAZZARO (ITALY),
HONORARY MEMBER OF THE AMERICAN VETERINARY ASSOCIATION,
PRINCIPAL AND DEAN OF THE ROYAL VETERINARY COLLEGE, LONDON

SUB-EDITOR:

GLADSTONE MAYALL, M.R.C.V.S.

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The Veterinary Journal.

AUGUST, 1934.

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THE PROGRESS OF VETERINARY SCIENCE.

THE articles published in the current issue of THE VETERINARY JOURNAL give some idea of the progress being made by veterinarians in the investigations into disease problems. It is true that within recent times public attention has been focussed on some of the major problems in the control of diseases of animals, especially those with a bearing on the welfare and health of human beings. Great as these problems are, and no one can deny their importance, it must always be kept in mind that diseases of all classes of domesticated animals come within the purview of veterinarians and, further, that often the detailed results of the investigation of diseases of one class of animal may have far-reaching effects in their application to disease problems of other animals. The "pure" laboratory worker can often by his patient researches throw light of much value on problems far apart from those engaging his attention. Diseases of sheep, poultry, dogs and cats, etc., must not be neglected, in spite of the fact that there exist diseases among our larger animals which are not yet thoroughly understood. The veterinary profession is, and must always be, comprehensive in its undertakings, and the owner, breeder and rearer of the sheep, fowl, dog or cat have claims on the veterinary profession just as has the general public, the care of whose food and milk supply must be in our charge. We are apt to become impatient with ourselves when we consider the enormous field that has to be covered; but a review of what we have

done and are doing will always reassure us that we are surely and steadily gaining a more intimate knowledge of the nature of the various disease problems among the domesticated animals.

The articles contained in this number show that we are progressing all the time. As more and more money is devoted to research, and as more trained workers become available, the progress will inevitably increase, for it is not difficult to grasp that the various researches are progressing on the right lines. The results of actual research must be converted into practical application. This is a subject requiring special attention all the while, for the end and object of all research must be the elaboration of practical methods for preventing and curing disease. It is very evident that all types are necessary : the research worker, those who devote their energies to the translation of the results of research into methods of practical application, and those who actually carry out the methods of control.

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General Articles

THE ERADICATION OF BOVINE CONTAGIOUS ABORTION.

By A. W. STABLEFORTH, D.Sc., M.R.C.V.S., D.V.S.M.

Research Institute in Animal Pathology, Royal Veterinary College, London.

THE purpose of the present article is to give briefly the present state of our knowledge regarding this preventable disease, and to outline the measures which may be taken for its control and eradication.

The reason for putting it forward at this time is, firstly, the numerous occasions on which we are asked for advice by veterinary practitioners; and, secondly, the evident and continuously increasing interest shown by stock owners themselves in ridding their herds of abortion, as is indicated by the number of meetings which they are holding up and down the country solely for the purpose of its discussion. Added impetus cannot fail to be given to this movement by the recent report of the Cattle Diseases Committee of the Economic Advisory Council, a report which should be in the hands of every veterinary surgeon.

There is general agreement that of the diseases which harass the cattle owner to-day, contagious abortion, due to *Brucella abortus*, causes him the most serious loss. On a large number of farms it imposes a continuous drain on an already precarious income, and in many cases undoubtedly makes all the difference between an annual profit and a loss.

Incidence and Economic Importance.—Although exact figures are not available for the country as a whole, the incidence of the disease appears to be increasing, and the average percentage of infected cows is at the present time generally agreed to be not less than 20 per cent., and is often put at 30 per cent. The average percentage of infected heifers is notably smaller, probably not more than 4 to 6 per cent. Not more than 10 per cent. of adult bulls are infected. The figures for first-herd tests of over 2,000 animals in unvaccinated herds carried out at this College, mostly during the last year, in which the whole milking herd was tested previous to the institution of an eradication scheme, showed from 12 to 70 per cent. of infected animals.

The primary loss, that of the calf, may not be of great importance in an ordinary milking herd, but in pedigree herds and where efforts are being made to grade up generally, the loss of a few calves may well nullify the work of years.

In the ordinary milking herd, on the other hand, the most serious economic factor is the definite loss of milk which occurs. As regards the loss in the individual, the earlier in pregnancy the abortion or premature calving occurs, the greater is

the loss. In addition to this, however, there is reason to believe that there is a definite decrease in yield in infected animals which do not acutally abort.

Estimates of the average loss vary, but comparisons, extending over a number of years, between the milk records of otherwise similar abortion-free and abortion-infected herds, and between infected and non-infected animals in the same herd, have shown that the average financial loss per cow per year as the result of abortion infection is not less than £10 per head. It is also an undoubted fact that failure to conceive or permanent sterility are more common in abortion-infected herds, whilst retention of fœtal membranes is a further source of trouble.

Contagious abortion is without question a cause of serious loss in several ways, and it is the opinion of many dairy farmers that, taking into account the uncertainties of being able to keep up a milk contract, and the general interference with farm routine and the regular spacing of calvings, that the total loss should be put at a considerably higher figure than £10 per head.

As an instance of the advantage of freedom from abortion, it has recently been shown from records of 3,510 adult animals which were under supervision for periods up to seven years (and covering in all 5,074 cow years) that, assuming the normal number of calves to be one per twelve months (and calling this 100 per cent. reproductive efficiency), non-reactors showed a reproductive efficiency of 87 per cent., with an abortion rate of 2 per cent.; whilst recently infected reactors had a reproductive efficiency of 55 per cent., and an abortion rate of 24 per cent.; and chronic reactors (*i.e.*, those of more than two years' standing), a reproduction efficiency of 76 per cent., and an abortion rate of 9 per cent.

Owners who rid their herds of contagious abortion at the present time will reap a considerable benefit later, when the establishment of clean herds has become more general, with the result that there is a premium on animals from herds which are known to be abortion-free, and also from the fact that they have a surplus of young stock from which they need only keep the best.

Measures which have been Proposed for Control.—The measures which have been proposed for dealing with contagious abortion fall under two main heads: namely, vaccination and actual eradication. It has also been suggested as a practical measure that the disease may be allowed to run its course. Such a policy, however, is necessarily costly, because a heavy loss is almost certain until the infection has spread through the herd; moreover, although after a storm of abortions there is usually a lull, this is dependent on the fact that the majority of the animals have become infected; and it offers no security to younger animals drafted into the dairy herd with their first calves or purchased from outside. They are, on the contrary, exposed to a severe risk of infection and consequent abortion. The actual abortions may decrease for several years, but the disease does not die out, and commonly flares up later. The policy of " doing nothing," when reduced to its lowest terms, means nothing more than casual vaccination, uncontrolled as regards dose or time, and must, therefore, be inferior to a regular policy of vaccination or eradication.

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Vaccination has been carried out with both dead and living culture. There is no evidence that vaccination with dead organisms is of any value. There is undoubtedly reason to claim that vaccination with living cultures does on the whole reduce the number of abortions, but its results vary markedly from herd to herd. In assessing its value, the fact that a period of numerous abortions is usually followed by one in which fewer occur should not be overlooked. Many of its reputed successes are due to this fact alone. Vaccination with virulent cultures also has the disadvantage that every animal must be injected, and that it must be repeated yearly. Moreover, if the best results are to be obtained, it is not altogether such a simple matter as is frequently thought, because there is ample evidence that it should be carried out at a definite period, one or two months before service. Vaccination with a living culture is nothing more nor less than a process of artificial infection at a time when the animal is insusceptible to actual abortion ; and this being so, it is frequently followed by many of the same undesirable sequelæ as natural infection, namely, sterility, retention of fœtal membranes and infection of the udder. Vaccination means, therefore, that every animal in the herd is artificially infected, and it can have no place in any attempt to eradicate the disease.

It may logically be laid down that resort should not be made to vaccination until a blood test has been made of the herd in order to find out how many animals are infected, and that it should not be used where it is at all practicable to sell reactors—if they are few or of no particular value—or to segregate them. It is for obvious reasons improper to infect a whole herd by vaccination when it is possible that only a small proportion of animals are infected and the eradication of the disease a simple matter. The ease with which contagious abortion can be eradicated by separation of infected and uninfected animals varies from farm to farm, and is, of course, much simplified by the existence of two farms or two sets of premises. But it should be emphasised that on a large number of farms where perhaps at first sight the two-herd system may appear to be an impracticable policy, a short time spent in considering the houses and pastures available will reveal a practical and practicable method of separation involving little expense. The precautions to be taken are few and simple, not costly, and not difficult to fit into ordinary farm routine ; and in any case will be amply repaid by the increased efficiency of an abortion-free herd.

Vaccination in its present form should be confined to herds which are not only badly infected, but where facilities for isolation are non-existent, or to those which are chiefly maintained by outside purchase. Efforts have been and are being made to prepare an efficacious vaccine which has none of the drawbacks of the present living vaccine, but there does not at present appear to be any prospect of attaining this object.

Epizootiology.

Although the epizöotiology of contagious abortion is a subject with which veterinary surgeons are familiar, it may not be out of place to recall the

main facts on which the policy of eradication now adopted in various countries is based.

More than 90 per cent. of abortions in cattle are of the contagious variety caused by *Brucella abortus*. A few are caused by other bacteria or by accidents. It is for this reason important that where an abortion occurs on a believed clean farm, immediate steps should be taken to ascertain if it is due to *Brucella abortus, i.e.*, to the contagious variety, or due to another cause.

Bovine animals are the only animals commonly affected. Brucella infection has, it is true, been demonstrated in other animals, but as far as this country is concerned, there is no reason whatever to believe that they are a common source of danger at present. Both male and female animals are susceptible at all ages, although the susceptibility may vary from time to time, and is believed to be higher in pregnant cows. Abortion most frequently takes place between the fourth and seventh month, but may occur at any time during gestation. Early abortions are often overlooked, the return of the animal to œstrum being put down to another cause. Similarly, premature calving is often put down to causes not in any way connected with contagion. It is, nevertheless, an established fact that the majority of premature calvings are actually due to Brucella infection. Dead calves born at full time are also more frequent in infected than in non-infected herds.

Infected animals by no means always abort, and most animals which have once aborted acquire a tolerance to the disease, with the result that they carry their next calf to full time. A small number of animals abort a second time ; rarely, three or more abortions will occur in the same animal.

The actual result of exposure to infection depends on three things : the individual resistance of the animal, the heaviness of the initial infection, and the time at which the animal is exposed to it. A severe exposure during the early months of pregnancy will almost certainly result in abortion. On the other hand, an exposure before conception will frequently lead to the development of tolerance before the period of susceptibility to actual abortion arrives, and consequently to the production of a normal full-time calf.

The important fact to be grasped is that animals which become infected are a danger to other cattle, whether they abort or not. Particularly is this so at calving time. The infected animal which calves at full time expels large numbers of abortion bacteria with the membranes and discharges, and is no whit less dangerous than the animal which actually aborts. The calf also is infected and infective.

Infection is much less commonly found in males and heifers than in cows. The reason for this may be a difference in susceptibility, but is in all probability found in the circumstances that calves, bullocks and heifers are generally kept apart from the older cows. Calves can and do become infected, but nearly always get rid of the infection within a few months of weaning. Thenceforward, under the system of management which is usual in dairy herds, they are kept apart, often

looked after by a separate set of attendants, and are therefore not exposed to much risk of infection until they are added to the main herd after the first calving. This alone is responsible for the fact that abortions are more frequent in the second pregnancy than at any other time. Heifers that do become infected during their first pregnancy nearly always calve prematurely or abort, often at an early stage of gestation.

Method of Infection.—Until the last few years it was believed that infection was almost invariably contracted by the mouth, *e.g.*, by licking infected animals which had recently calved or aborted, by licking or eating an aborted fœtus, or by ingestion of food contaminated with the discharges from an infected animal; or, in the case of calves, by the ingestion of infected milk. There is no doubt that these are still common methods.

It has, however, now been shown both in guinea-pigs and in cattle, that animals may be readily infected *via* the conjunctiva; or, with rather less certainty, through the broken or intact skin. One set of workers has recently reported that out of seventeen cattle which had been exposed to infection by dropping abortion organisms into the eye, sixteen became infected, and that the dose required to produce infection by this route was surprisingly smaller than that required to infect by the mouth. The same workers succeeded in infecting four out of seven heifers by applying abortion organisms to a small area of unbroken skin in the gluteal region, and each of two heifers whose skin was first shaved and lightly scraped. Due precautions were taken to avoid infection by any other route than the skin of the area treated.

The actual importance of these latter methods in practice is difficult to assess, but it is not unreasonable to suggest that they are probably important. They do not alter in any way the methods which should be adopted to eradicate the disease, but serve further to emphasise the necessity for taking adequate precautions wherever infective discharges or milk are concerned. There is as yet no evidence that abortion infection can be carried from one cow to another by the milker's hands, but it is a probability which should not be overlooked.

Incubation Period.—The period which elapses between infection and abortion is variable, and in fact infected animals may not abort. Most abortions probably occur within two to four months of infection. The period between infection and the appearance of specific antibodies in the blood is probably not usually longer than one month.

Organs Infected and Methods of Spread.

In the Adult Female the causal organism occurs in the pregnant uterus and in the udder and associated glands. The fœtus, fœtal membranes, uterine discharges and milk are thus all infective. In the non-pregnant animal its predilection seat is the udder and its glands, where it causes a mild chronic mastitis of a non-clinical character. When pregnancy occurs the causal organism migrates to the uterus, where it remains until after parturition.

In the Adult Male it is found in the generative organs, and may give rise to orchitis, in which case the semen is infective. The testicles gradually become enlarged and firm.

Joints.—Commonly the knee and hock may also be infected. Many of the hygromas of cattle are of this nature.

In Calves the causal organism is present in the intestines and internal organs. Their fæces are thus infective. The skin of calves is, of course, also contaminated at birth.

The period when danger of spread to other animals is at its greatest is calving time. The causal organisms are present in the membranes and discharges of infected animals in enormous numbers, and it is necessary to take the most strict precautions against infection of other animals from these sources. It is to be emphasised, furthermore, that this is so whether the fœtus is aborted or whether a full time normal calf is produced. The false feeling of security engendered by a normal calving is doubtless responsible for much spread of the disease.

Persistence of Infection.—Uterine infection does not persist for long after parturition: rarely more than three weeks to a month. The udder, on the contrary, usually remains infected for a long period, often for life. Animals which have once been infected become, therefore, in a large majority of cases, permanent carriers. A few animals recover, but no use can be made of this in practice, because it is impossible to foretell the result in the case of any particular animal. The length of survival of *Brucella abortus* outside the animal body is small, but varies according to the conditions. It is quickly killed by direct sunlight, drying, and by weak disinfectants or soda. On pastures or other contaminated materials it is seldom alive after three months, and is usually dead within a shorter period.

Calves.—It is an established fact and of outstanding importance in the control of contagious abortion in the field, that calves are relatively resistant until the age when the reproductive functions mature, *i.e.*, towards the end of the first year. Calves born from infected mothers are infective, and carry the causal organism in their intestines and internal organs of birth; but, if fed on uninfected milk, most of them get rid of the infection within a few months. It is believed, however, that a small proportion become permanently infected. The fact that calves from infected mothers do carry the infection should be borne in mind, because it is possible that they may be the means of introducing infection into a clean herd or of infecting calves from uninfected mothers by way of their fæces.

Calves which are fed on milk which contains *Brucella abortus* (*i.e.*, from infected cows), carry the infection until such time as the uninfected milk is no longer used, but usually free themselves of the infection within a few months after its discontinuance. Calves should preferably be fed on milk from uninfected animals, or, if milk from infected animals is used, it should be scalded.

Whatever method of feeding is adopted, however, calves form the mainstay of every effort to build up a herd free from contagious abortion (as, indeed, they do of attempts to deal with all of the major diseases of cattle). They should

have no contact with infected animals after the age of six months, and preferably three months. Beef herds, of course, raise a problem in this connection, but must be dealt with according to circumstance by the use of uninfected mothers only.

Bulls.—It is improbable that the bull plays any large part in the dissemination of contagious abortion. Bulls that are infected in the genital organs excrete the organisms in the seminal fluid, and for this reason bulls that react should not be used. A bull may also convey infection mechanically, by serving a healthy cow shortly after an infected one. The period during which the genital passages of an infected cow actually contain the *Brucella abortus* is, however, limited: probably never more than one month; and danger from this source can be avoided by never having cows which have aborted, or which are reactors, served until at least one, and preferably two, months after parturition. The bull will thus also be protected. As a further precaution, the bull's abdomen and prepuce may be washed after serving a reactor, and at least twenty-four hours allowed to elapse after serving a reactor before the bull is used for a clean animal. Precautions should also be taken against infection of bulls by not pasturing them with infected animals. Where it is possible separate bulls should be used for healthy and infected animals.

Diagnosis.

From what has already been said, it follows that in any attempt to eradicate contagious abortion it is necessary not only to deal with those animals which actually abort, but all which are infected with the causal organism, *Brucella abortus*.

Fortunately, the blood serum agglutination test provides a satisfactory way of doing this, and in competent hands is a method of accuracy. Like other serological methods it is, of course, not a hundred per cent. accurate; there is always an incubation period, which may vary from a week to a month, or in rare cases longer, between the actual infection and the time when the blood sample gives positive evidence of the disease; and occasionally an animal will not react until several days after abortion or calving. There is, however, no longer room for doubt that the test is accurate in the vast majority of cases, and that periodic testing, combined with selling or segregation of reactors and improved hygiene, is an effective way of eradicating infection. Most herds, even badly infected herds, can be put on a satisfactory breeding basis within twelve months.

Many animals which react to the test do not abort, but it has already been shown that these are not less dangerous than those which do in fact abort.

In any large number of blood tests there are always one or two animals which give a borderline reaction which can only be interpreted as "doubtful" or "suspicious." Such reactions may mean that the animal is in the early stage of an infection, and that it will react definitely a week or two later, or may indicate a slight infection from which the animal will recover. A few animals continue to give a doubtful reaction for several tests. It is probable that most of these

doubtful animals have actually been infected ; doubtful results are not common in clean herds or in animals on infected farms, *e.g.*, heifers which have not been in contact with infection. These doubtful animals are in most cases not dangerous from the point of view of dissemination of the disease at the time ; but if pregnant, should be isolated or carefully watched for impending abortion, and should be re-tested in two or four weeks, when the true state of affairs can usually be decided.

Cows which have once given a positive reaction nearly always continue to do so, and there is general agreement that no useful purpose is served by re-testing them.

Virgin heifers which give a weakly positive reaction may recover ; but animals which have been served nearly always become permanently infected.

Frequency of Testing.—Since the incubation period is commonly from one to four weeks and sometimes longer, it is not to be expected that one test will detect all the infected animals. In practice it generally picks out 80 to 90 per cent. It is, therefore, advisable to make the first two, or possibly three, tests at short intervals, one to two months, particularly if the incidence of infection is high or there is evidence of an active infection (a relatively large number of abortions) or recent spread.

Subsequent re-tests should be made at intervals of three or four months during the first year, after which the interval should be increased according to the degree of separation of reactors and non-reactors, number of outside purchases, and results obtained. Where two or three tests are carried out at intervals of about a month, and reactors sold or put on another farm, there should be no difficulty in getting rid of the infection quickly. Where reactors have to be retained on the same premises the process of eradication is usually a gradual one.

Prompt Diagnosis.—The importance of a prompt diagnosis of the first case of contagious abortion on a farm cannot well be over-emphasised, because the time which it will take to eradicate the disease and therefore the cost and inconvenience to the ower are largely dependent on it.

When an abortion occurs, the animal should be immediately and strictly isolated in a box, and a blood sample should be taken at once and sent for agglutination test. The aborted calf in the place where abortion has taken place should also be suitably dealt with at once. A blood sample should also be taken from any animal which calves prematurely. Prompt action will amply be repaid by the prevention of further spread thus attained. Abortions in previously clean herds are frequently put down to accidents, with disastrous results to the rest of the herd, and veterinary practitioners can be of inestimable benefit to farmers by inculcating the elements of prevention in clients who would otherwise neglect the obvious means of avoiding a serious spread of disease.

If an abortion or premature calving is found by the agglutination test to be of the contagious variety, a test of the whole herd should be made at once. Many an outbreak can thus be nipped in the bud, and the difficulties of dealing with a badly infected herd avoided.

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Control.

The method of control now generally practised in this and other countries is dependent on the separation of animals which react to the agglutination test (infected animals), from those who do not react (uninfected animals), and the replacement of infected animals by uninfected young stock bred on the farm or purchased outside, when possible, from another abortion-free farm. This method is applicable to herds which are wholly or largely maintained by home-bred stock —self-contained herds. It can only be carried out with difficulty where numerous outside purchases are made, and cannot at present be applied to those herds which are chiefly maintained by outside purchase—sometimes called " flying herds." Fortunately it has been estimated that at least 30 per cent. of the herds of the country are sufficiently self-contained to make eradication practicable. When a large proportion of these have been freed from infection it will be possible to deal with the herds which are recruited largely by outside purchases.

The eradication of contagious abortion from herds which are self-contained, or largely so, presents no technical difficulties. Practical difficulties there are, chiefly owing to the necessity for keeping up milk contracts and the consequent purchase of cattle from outside; but the disease can quickly be got rid of by any owner who, realising the cost of an infected herd, makes up his mind to follow a definite policy and is willing to carry out regular tests of all his cattle and to take certain definite precautions.

The time which complete eradication of the disease will take is dependent on the initial incidence of the infection, the degree of separation possible, and the care taken to prevent dissemination of infection. Where the owner has two farms and can place all the infected stock on one farm, with separate attendants, utensils, etc., the eradication of the disease presents no important difficulties. No greater difficulty should be experienced where, owing to the fact that few animals are affected or because the infected animals are of little value, they can be disposed of at a small loss. In either of these cases there is no doubt that the immediate disposal of reactors or removal to one farm is the only sound policy. Farms of these two classes are, however, admittedly not a large proportion of the whole.

Eradication by blood testing and segregation of reactors can, however, be confidently recommended on farms where conditions are not so ideal. The farm which commonly comes up for consideration by the veterinary practitioner is the farm carrying 30 to 100 head, where two or more sheds are available, a certain number of calving boxes, or sheds which can be adapted as such, and reasonable pasturing amenities. In these cases reactors and non-reactors should be housed in separate sheds or yards, grazed on separate pastures, provided with a separate water supply, and, when practicable, looked after by separate attendants. Where separate attendants are impracticable, great care should be taken that infection is not spread by hands, boots and clothes of attendants, or by means of feeding and other utensils. If reactors and non-reactors can be brought into their

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respective sheds by different routes it is an advantage, but it is not a necessity. Separate pastures are essential, and some difficulty may be met with in regard to these, and also in regard to watering facilities. A short time spent in considering the facilities available frequently shows that virtually separate herds can be established on one farm. A road or lane, a stream, a succession of arable fields, a wood, a really thick hedge, can often be used to separate reactors and non-reactors. Failing such natural boundaries, a double fence can be erected at no great cost with stakes and wire.

The difficulties are greater where only one shed is available for both reactors and non-reactors ; elimination will naturally be slower and the period of two or three monthly tests will be necessarily prolonged. But in this case, although the chances of spread are clearly greater, experience has shown that a great deal can be done provided regular testing is carried out and the simple principles on which eradication is based thoroughly explained to all those on the farm. The disease can be prevented from spreading, and the number of new infections gradually decreased until sufficient healthy young stock are available to replace the older infected cattle. In the case of these one-shed farms, the usual policy is to put all of the reactors at one end of the common shed, nearest the drainage outlet, and to have an empty standing or two between reactors and non-reactors. Non-reactors must always be fed and milked before reactors, and walking to and fro from one to the other should be avoided as far as possible. A trough of any good disinfectant placed between the two and at every entrance to the clean position of the shed will help to prevent infection being carried on boots, and what is more important, will serve as a constant reminder to all that the non-reacting section is to be jealously guarded from infection from the reactors. Wherever possible a temporary partition should be put up between the infected and uninfected sections of the herd.

Manure from reactors or from calving boxes should be used for arable land or for grassland kept for reactors; but where it is desired to use manure for grassland used by the clean herd it should be stacked for three months. It should always be taken well away from the sheds, and manure heaps should be fenced, unless so placed that non-reactors can have no access to them.

Pastures which have been grazed by the infected section of the herd may safely be used for clean animals after three months have elapsed.

Nutritional Influences.—It is often suggested that the feeding of a ration deficient in mineral, or low in protein, may lower the resistance of cattle to contagious abortion. This suggestion has recently been tested in an experiment which was carried out on forty-four young animals of the same breed, which were under observation for a period of five years. Two main groups were made, one of which was given a " good " ration containing ample minerals, cod-liver oil and iodised salt ; the other a " poor " ration (though one commonly fed), which was low in lime, somewhat low in protein, and had only a moderate phosphorus content. No evidence was obtained that the difference in the diet had any influence on the occurrence of abortion.

Calving Boxes.—Calving is the time of greatest danger, and when spread of infection generally occurs.

Both infected animals which abort and those which calve to time are dangerous, and excrete, literally, millions of infective organisms, immediately before and for at least a week after the act of abortion or calving. This fact cannot be stressed too much. If adequate precautions are taken only at calving time a great deal of dissemination of infection can be avoided. The danger from this source gradually decreases, and after two or three weeks no longer exists.

In order to prevent the spread of infection at this period, use should be made of calving boxes. Where sufficient are not already available, quite satisfactory calving boxes can be made by dividing up a shed or stable by means of temporary partitions. All calving boxes should have an impermeable floor and walls which can be easily cleansed and disinfected.

When sufficient calving boxes can be provided for both reactors and non-reactors, all animals should be isolated at calving time. If there is a shortage, the best use to be made of them will depend on whether reactors or non-reactors are in the same shed or not. Where reactors and non-reactors are in the same shed, it is essential that the available boxes should be used for reactors. These must never be allowed to calve in the common shed. When, on the other hand, the reactors are stalled in a shed by themselves, and thus isolated, they may be allowed to calve in it, and the calving boxes reserved for the clean herd. The reason for this is that further spread of infection in the infected sections of the herd is of little or no importance, whereas in the clean herd it would be serious. Most animals in the clean herd will calve normally ; the object of isolating them is to prevent the possibility of spread from the occasional animal which has become infected but which does not react till a week or two after calving or abortion.

Animals should be moved to calving boxes a week before calving is expected, or at once if any suspicion of abortion or premature calving is seen, and they should be kept isolated for two or three weeks until all discharge has ceased, and preferably until after a negative blood test. Particular care should be taken to cleanse and disinfect calving boxes after they have been used for reactors.

Outside Purchases.—The purchase of adult animals in the open market or from herds of unknown history is the way in which infection is nearly always brought into a herd, and should be avoided whenever possible. In most herds, however, some outside purchases are inevitable, if the milk yield and the efficiency of the herd is to be maintained. Animals from abortion-free herds are naturally to be preferred, but it may not be possible to buy these owing to their cost, or the shortage of supply. Purchase should be made subject to test when practicable, but when no test is made before purchase, animals should be quarantined until they have passed one blood test, or, better, two tests with an interval of one month. When pregnant animals are bought, it is essential that they should be strictly

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isolated until two to three weeks after calving, and until they have passed a blood test at the end of that time.

The actual class of animal purchased will vary according to the farm or type of business carried on. The safest animals to buy are non-pregnant heifers or cows. If heifers are bought there is less chance of disappointment, because the majority will be non-infected. A further advantage is, of course, that the loss is usually smaller if they have to be resold.

Calves are also safe purchases provided they are, as is usual, kept away from adult animals. The most dangerous animals to buy are, unquestionably, pregnant cows, and to a less extent pregnant heifers.

The actual conditions vary much from farm to farm, and it is here that the practitioner with his knowledge of the epizöotiology of the disease can be of service to the client by adapting the main principles of eradication to a practicable and efficient scheme for a given farm.

Identification and Records.—Where abortion is being eradicated by blood tests and segregation, it is essential that each animal shall be permanently identified. The best way is by horn or ear number.

Samples of blood for agglutination test should bear the permanent identification mark or name of the animal. This enables the laboratory doing the blood testing to keep a record of each individual animal's history, and such records may be of great help in deciding whether a certain animal can be passed.

It is also strongly to be recommended that veterinary practitioners who are responsible for an eradication programme should themselves keep a record of each animal in the herd filed under the animal's permanent identification mark, and comprising a record of the date and result of each test, together with the age, calving and service date and history of the animal. In this way he can see at a glance the status of each animal, and in conjunction with the owner decide on future policy in the herd. Such records are at present usually kept by the laboratories that do the tests, but will obviously become increasingly difficult as more and more herds are examined. Quite apart from this, recording falls properly within the field of the practitioner, who not only adds to his own interest, but can also, by keeping them, be of much greater service to his client. Further, the keeping of close records by his veterinary adviser will often encourage an owner to take much greater pains than would otherwise be the case. If the practitioner feels, however, that records are best kept by the laboratory concerned, and it is essential that records are kept, then care should be taken that all relevant information is supplied promptly. Only in this way can accurate, and therefore useful information, be collected in regard to the problem of eradication in general.

We have already sufficient facts on which to say definitely that contagious abortion can be and has been eradicated by segregation of reactors. There are still gaps in our knowledge which can only be filled by a concentrated effort by all concerned. In this the man in the field can contribute no small share.

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LAMB DYSENTERY IN NORTH WALES.

By R. F. MONTGOMERIE, B.Sc., Ph.D., F.R.C.V.S., and W. T. ROWLANDS, M.R.C.V.S., D.V.S.M.

School of Agriculture, University College of North Wales, Bangor.

SINCE Gaiger and Dalling (1921)[6] published the account of their early investigations into bacillary dysentery in lambs, the disease has appeared in, or spread to, counties far distant from the original border area. It was first diagnosed in North Wales in 1926, when serious losses among young lambs were encountered in one district. Since then it has been shown to exist in several other localities, and year by year each infected area has materially increased in size.

There is no evidence that the appearance of the disease in any of the North Wales areas had any connection with the original centres of infection in the North, and only in some instances could it be traced to association with existing local centres. Not uncommonly the practice of purchasing " couples," ewes with young lambs at foot, or of off-wintering ewe lambs, some of which returned to their grazings advanced in pregnancy, has been responsible for the introduction of infection into clean areas. In other instances the most thorough inquiry has failed to elicit information which could suggest a route of infection.

One owner contended that a serious diarrhœic disease of lambs existed many years ago, and that lamb dysentery was present on his farm when the present tenancy commenced in 1917. Apart from that record, it would appear that the disease made its appearance, or began to make its presence felt, in the centres first affected about 1920. A careful review of all the information gleaned during eight years contact with the disease position in North Wales almost forces one into the belief that the causal agent is a recent introduction into the province or that some change in the management of the sheep or their grazings has afforded a pre-existing infectious agent opportunity to set up disease.

Gaiger and Dalling originally believed[6] that the disease was caused " by a bacillus of the coli type which invades the body from the inflamed bowel," but in a later note (1923)[7] expressed the view " that an anaerobe of the *B. welchii* type and a bacillus of the coli type acting conjointly are responsible for the production of lamb dysentery." In this communication they reported the first successful attempts to reproduce the disease in young lambs by feeding the intestinal contents of naturally-infected lambs, and by feeding a mixture of cultures of this anaerobe and *B. coli* isolated from naturally occurring cases of the disease.

Dalling (1926)[2] reported his reproduction of the disease in lambs up to twelve hours old by feeding intestinal contents, ground-up ulcers, etc., from hyperacute field cases. He consistently reproduced lamb dysentery by the simultaneous feeding of cultures of strains of a Welch-like organism and of *B. coli* isolated from field cases, but could only reproduce it by feeding cultures of the anaerobe alone,

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when the organism was " in an appropriate condition of pathogenicity." The inoculation of antiserum prepared against this strain of *B. welchii* prevented the reproduction of the disease by these and other means, whereas the inoculation of *B. coli* serum and of normal horse serum had no such influence. With his collaborators, Mason and Paul, he put forward striking evidence of the efficiency of prophylactics prepared from his strains of *B. welchii* and *B. coli* in the prevention of lamb dysentery in the field.

In 1927, Dalling, Mason and Gordon[4] reported further observations on the prevention of lamb dysentery in the field. One of the present writers (R. F. M.) had the privilege of collaborating in some of their field trials, which demonstrated that " vaccines made from a type of *B. welchii* and from strains of *B. coli* isolated from lambs affected with lamb dysentery " had considerable power in protecting the lambs of vaccinated ewes, but that the *B. coli* part of the vaccine played little or no part. This latter contention received full confirmation from observations on the value of the injection of young lambs with serum prepared from the anaerobe alone.

Dalling's observations led him in 1928[3] to express quite confidently the view that this Welch-like organism was the exciting cause of lamb dysentery, which view he based very largely on the work reviewed above and on further experience of the successful field use of prophylactics prepared from it alone. In this communication he pointed out certain striking differences between his " L.D. bacillus " and *B. welchii*.

In 1928, Pool, Preston and Brownlee[16] reported a series of attempts to set up the disease experimentally by various paths of infection, all of which gave negative results except the ingestion of fæces from natural cases by newly-born lambs, in which case a positive result was obtained in four out of seven experiments.

The successful use of prophylactics prepared from the " lamb dysentery bacillus," was further reported upon by Dalling, Mason and Gordon (1928),[5] who found that the addition of a killed suspension of *B. coli* to toxin-antitoxin mixture of the lamb dysentery bacillus did not enhance the value of the latter as a prophylactic by means of ewe inoculations. Toxoided whole culture of the anaerobe was successfully employed for this purpose. Furthermore, lamb dysentery bacillus antitoxin, when injected into young lambs, gave a degree of protection approximating to 100 per cent. Parallel results were obtained in North Wales.

So far as the writers are aware, no further work on the experimental reproduction of the disease has been reported, and the continued successful general application of prophylactics prepared from the lamb dysentery bacillus came to be accepted as proof that this organism is the cause of lamb dysentery, without evidence as to the manner in which it set up the disease. Dalling (1928)[3] recorded that in only twelve of the many cases investigated during the course of four years was he able to isolate the lamb dysentery bacillus from the tissues of lambs killed while showing symptoms of dysentery, whereas it was abundantly present in the intestinal contents, even when there was no evidence of ulceration. It

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appeared to the writers that lamb dysentery might well prove to be an entero-toxæmia, and since 1931 they have tested for toxicity the intestinal contents of the cases they were able to obtain. The general and very successful use of lamb dysentery serum in North Wales made it difficult to obtain material in 1931 and in 1932. In each of these seasons six cases, mostly obtained from fresh outbreaks of the disease, were investigated. In the spring of 1933 a new position arose, in that on certain infected farms inoculation of the lambs with serum within twelve hours of birth failed in a varying degree to control the disease, whereas in previous years it had afforded complete, or almost complete, protection. Furthermore, lamb dysentery appeared for the first time on certain farms which had a previous history of heavy annual losses from pulpy kidney disease in lambs over three weeks old. In these circumstances the writers had very largely to restrict their investigation to such outbreaks, and the intestinal contents of sixteen cases of lamb dysentery were dealt with.

TABLE No. I.

THE PROTECTION AFFORDED BY VARIOUS ANTISERA AGAINST TOXIC INTESTINAL CONTENTS FILTRATES. OBSERVATIONS DURING THE 1931 AND 1932 SEASONS.

	Case No.	Toxic dose of filtrate	Toxic dose of filtrate plus equal volume of antiserum.				
			B. welchii	L.D. bacillus	*V. septique*	*B. œdematiens*	*B. paludis*
1931	1	0.05	†	L	†	†	—
	2	0.05	†	L	†	†	—
	3	0.1	†	L	†	†	—
	4	0.05	†	L	†	†	—
	5	0.1	†	L	†	†	—
	6	0.05	†	L	†	†	—
1932	1	0.05	†	L	†	†	L
	2	0.05	†	L	†	†	L
	3	0.05	†	L	†	†	L
	4	0.05	†	L	†	†	L
	5	0.05	†	L	†	†	L
	6	0.1	†	L	†	†	L

† = Died. L = Lived.

The Toxicity of Intestinal Contents.

In obtaining a suitable filtrate of intestinal contents, the procedure adopted was that of expelling the ingesta into a sterile glass jar by lightly running the unopened small intestine between the finger and thumb, the addition of an equal volume of physiological saline, and shaking to thoroughly mix. The mixture was then centrifuged at 2,500 r.p.m. for half an hour. The resulting supernatant was roughly filtered, then drawn through a Seitz clarifying pad and finally through a Seitz filter fitted with an E.K. or E.K. Special pad. In this manner sterile filtrates were regularly obtained, although in some instances only in small quantity. The final filtrate was tested for toxicity by the intravenous injection of a test dose (0.4 c.c.) into mice 20 to 22 gm. in weight. Where the test dose proved

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non-toxic, the filtrate was discarded. Otherwise a dose which killed within a convenient period, one probably containing several or many minimum lethal doses, was selected for further work. No attempt was made to determine the minimum lethal dose.

Of the twelve cases obtained in the 1931 and 1932 seasons the lambs were less than five days old, and with one exception showed typical ulceration of the bowel. The exception, a 1931 case, was a three-days-old lamb in which there was only marked inflammation of the bowel wall. In each a highly potent intestinal toxin was readily detected (Table I).

Since special significance is attached to the investigation of the 1933 cases, a brief history of each flock and case is given :—

Case No. 973.—Flock affected with lamb dysentery for at least three years. Lamb three days old. The markedly inflamed bowel wall showed commencing ulceration.

Case No. 942.—Flock has long history of P.K. (pulpy kidney disease) losses. Twenty younger lambs had been lost this season. Lamb four days old, showing typical ulceration of the bowel wall.

Case No. 958.—Flock affected three years. Lamb six days old. Some inflammation of intestinal wall and distinct but small ulcers.

Case No. 991.—Probably first year of lamb dysentery. Lamb three days old. Typical ulceration of bowel wall.

Case No. 10.—Flock has a long history of losses typical of pulpy kidney disease. Ewe stock 100. 1932: twenty lambs under ten days and other fifteen under one month died. Practitioner diagnosed lamb dysentery. 1933: lambing commenced March 15th, and losses about March 30th. Then all lambs inoculated with lamb dysentery serum, and each lamb born thereafter inoculated at birth. This was the nineteenth inoculated lamb to die ; aged one week and inoculated at birth. Inflammation of wall of intestine, but no ulceration.

Case No. 22A.—Same flock as Case No. 10. Lamb five days old and inoculated at birth. Inflammation, but no ulceration of bowel wall.

Case No. 22B.—Same flock as Case No. 10. Lamb ten (?) days old. Not inoculated. Healing ulcers of intestinal wall.

Case No. 28.—Same flock as Case No. 991. Lamb four days old. Typical lamb dysentery ulceration.

Case No. 70A.—Flock has long history of heavy losses typical of P.K. Ewe stock 400. In 1932, 80 lambs were lost, mostly one month old, but owner thought not quite typical of P.K. A few younger lambs died towards the end of lambing. One of these showed typical lamb dysentery ulceration on examination at the laboratory. In 1933, no losses until earlier lambs were one month old. Then deaths suggestive of P.K. began, but the age at which lambs died became less, and two-weeks-old lambs died after showing symptoms suggestive of lamb dysentery.

The farm from which Case No. 958 was obtained sends its ewes to summer grazing common to this farm. This is the only possible contact with lamb dysentery infection. Eventually, among the 400 lambs born, 200 deaths occurred, this lamb being the one hundred and twentieth to die; it was about ten days old. There was evidence of scouring and much inflammation of the jejunum, which showed many small and one large ulcer.

Case No. 70B.—The one hundred and twenty-first lamb dead on the above farm. Two weeks old. One part of the jejunum ulcerated and a localised peritonitis causing adhesion of adjacent coils of intestine.

TABLE No. II.

THE PROTECTION AFFORDED BY VARIOUS ANTISERA AGAINST TOXIC INTESTINAL CONTENTS FILTRATES. OBSERVATIONS DURING THE 1933 SEASON.

		Toxic dose of filtrate, plus equal volume of antiserum.						
			Lamb dysentery bacillus					
Case No.	Toxic dose of filtrate	*B. welchii*	Q232	C1852	C1971 A	*B. paludis*	*B. ovitoxicus*	*B. welchii* (Wilsdon, type D)
	c.c.							
937	0.2 †3	†3	L	†4	†3	†3	L	L
942	0.2 †3	†3	L	†3	†4	†3	L	L
958	0.2 †4	†5	L	†4	†6	†5	L	L
991	0.2 †¼	†¼	L	L	L	L	†½	†½
10	0.2 †1½	†2	L	†1½	†1½	†1½	L	L
22A	0.4 †5	†4	L	†6	†5	†6	L	L
22B	0.4 †5	†5	L	†6	†6	†6	L	L
28	0.2 †¼	†¼	L	†½	†½	†¼	L	L
70A	0.2 †½	†2	L	†1½	†½	†2	L	L
70B	0.2 †2	†2	L	†2	†1½	†2	L	L
84	0.2 †¼	†¼	L	†½	†¼	†½	L	L
85A	0.4 †¼	†½	L	†½	†½	†½	L	L
85B	0.2 †2	†3	L	†5	†4	†5	L	L
85C	0.2 †¼	†¼	L	†½	†½	†1	L	L
87	0.2 †4½	†4½	L	†4½	†5	†5	L	L
90	0.05†¼	Not done: filtrate-serum mixtures turbid.						

† = Died (figures = interval in hours between inoculation and death). L = Lived.

Case No. 84.—Flock affected with lamb dysentery three years. Serum inoculation successful in 1931 and 1932, but quite inefficient in 1933. Lamb inoculated with serum two hours after birth; died seven days later. Typical ulcers localised to first portion of jejunal wall.

Case No. 85A.—Same flock as Case No. 70A. Lamb three days old. Typical lamb dysentery ulceration.

Case No. 85B.—Same flock as Case No. 70A. Lamb three weeks old. Inflammation of bowel wall.

Case No. 85C.—Same flock as Case No. 70A. Lamb ten (?) days old. Much gas in inflamed intestinal wall.

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Case No. 87.—Lamb dysentery not previously detected in flock, which, however, was a draft from a distant farm (in supposedly clean area) in the same ownership. Lamb nine days old; had shown symptoms consistent with lamb dysentery. Marked inflammation of small intestine and many tiny ulcers widely distributed throughout, together with an occasional larger ulcer.

Case No. 90.—Same flock as Case No. 87. Three-days-old lamb found dead. Typical ulceration throughout small intestine.

As is shown in Table No. II, a toxic filtrate was obtained from the intestinal contents of each of these cases.

The Nature of the Toxic Element.

Since Dalling (1928)[3] demonstrated that antitoxin prepared from the lamb dysentery bacillus neutralised the toxin of *B. welchii*, but that the converse did not hold good, other organisms bearing a similar relationship to *B. welchii* have been isolated, notably *B. paludis* (McEwen, 1929),[10] and *B. ovitoxicus* (Bennetts, 1932[1]). The interrelationship of the toxins of these organisms has been the subject of much study. Mason, Ross and Dalling (1931)[11] and Mason (1933)[12] concluded that the toxin produced by the lamb dysentery bacillus was the same as that produced by *B. paludis*, but Bennetts[1] observed that the lamb dysentery serum he had received from the Wellcome Laboratories in 1929 and the *B. paludis* serum supplied to him by McEwen behaved very differently when mixed with *B. ovitoxicus* toxin; the former neutralising, whereas the latter did not. Wilsdon (1931),[17] in a comprehensive study of the classification of *B. welchii*, showed that, according to the toxin they produced, all his strains fell into one of four groups, which he named *B. welchii*, types, A, B, C, D; the three former corresponding respectively to *B. welchii*, the lamb dysentery bacillus, and *B. paludis*. The fourth, type D, the writers believe to be identical with *B. ovitoxicus*.* He showed that although type B (lamb dysentery bacillus) toxin was neutralised by type C (*B. paludis*) antitoxin, it differed from the toxin of the latter in that its antitoxin neutralised the toxin of type D (*B. ovitoxicus*), whereas type C antitoxin did not. He therefore concluded that after eighteen hours' incubation type B toxin contained a fraction common only to it and to type D, but in concentration too low to be demonstrated by the methods employed. When the period of incubation was increased to three days this common fraction was readily demonstrable in type B toxin, in that type C antitoxin did not then neutralise, whereas a combination of types C and D antitoxins did.

From Bennetts' and Wilsdon's observations one would conclude that antiserum prepared from the lamb dysentery bacillus neutralises the toxins of *B. welchii*, the lamb dysentery bacillus, *B. paludis* and *B. ovitoxicus*. Gill,[8] however, noted a difference in the power of the two batches of lamb dysentery serum supplied to him to neutralise toxin of the *B. ovitoxicus* type. Montgomerie and Dalling[13] also noted a similar variation, and the writers have shown[15] that whereas the toxin in the intestinal contents of lambs dead of pulpy kidney disease was

*See footnote on page 329.

neutralised by serum prepared from *B. ovitoxicus* and from *B. welchii* (Wilsdon' type D), the available batches of lamb dysentery serum (all prepared at the Wellcome Physiological Research Laboratories) differed in this respect. One batch prepared prior to 1930 neutralised, whereas two of 1932 preparation did not. It was therefore concluded that the 1930 serum contained antitoxin capable of neutralising the toxin of *B. ovitoxicus*, whereas that of recent production did not.*

The nature of the toxic element present in the intestinal contents filtrates from these cases of lamb dysentery was investigated by mixing the " toxic dose " with an equal volume of the available antitoxins, incubating the mixtures for half an hour at 37° C. and then injecting them intravenously into mice. The mice were kept under observation for three days.

Since the findings of Bennetts and of Wilsdon did not become known until after the 1932 lamb dysentery season, antitoxin of the *B. ovitoxicus* type was not available for this work until 1933. In 1931, antisera prepared from *B. welchii*, the lamb dysentery bacillus, *B. œdematiens* and *V. septique* were employed, and in 1932 *B. paludis* antiserum was also used. These antisera were kindly supplied by the Wellcome Physiological Research Laboratories. From Table No. I it will be seen that in 1931 only lamb dysentery serum, and in 1932 only that serum and *B. paludis* serum, neutralised the toxic element.

In view of these results, only antisera prepared from members of the Welch group were used in the 1933 neutralisation observations. The following, as designated by the indicated source of supply, were available: (1) " *B. welchii* serum G.G., C2748A," Wellcome Physiological Research Laboratories; (2) " Lamb dysentery serum, Q.232," issued by Burroughs Wellcome & Co., for field prophylaxis, in 1929; (3) " Lamb dysentery serum, C1852," W.P.R.L., produced in 1932; (4) " Lamb dysentery serum, C1971A," W.P.R.L., produced in 1932; (5) " *B. paludis* antiserum," W.P.R.L.; (6) " *B. ovitoxicus* antiserum No. 3," Bennetts; (7) " *B. welchii* serum, anti-Wilsdon's type D," W.P.R.L.

Table No. II sets out in detail the observations made on the neutralisation of the 1933 filtrates with these antisera. The *B. welchii* antiserum failed to neutralise the toxic element in any case; the lamb dysentery serum Q.232 neutralised in every instance; the lamb dysentery sera of 1932 production and the *B. paludis* antiserum neutralised in one instance only, and the *B. ovitoxicus* antiserum, like that prepared against Wilsdon's type D, neutralised in every instance except that in which the recently produced lamb dysentery serum and the *B. paludis* antiserum neutralised.

These results further demonstrate that the lamb dysentery sera of recent production contained no antitoxin capable of neutralising toxin neutralised by

* Wilsdon has recently shown that on the results of cross-immunity experiments, *B. ovitoxicus* is apparently identical with his *B. welchii* (type D). (Univ. Camb. Inst. Animal Path., Third Report, 1933, pp. 46–51.)

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B. ovitoxicus and *B. welchii* (Wilsdon's type D) antisera, and before proceeding to type the toxic element in the filtrates obtained during the three seasons it is necessary to consider the antitoxic content of the lamb dysentery serum used in 1931 and 1932. Since the serum used in 1931 failed to neutralise the toxic element of a pulpy kidney filtrate[14] it may be assumed that it contained no antitoxin of the ovitoxicus type. The serum used in 1932 did, however, appear to partially neutralise pulpy kidney intestinal filtrates.[15] It may have contained at least a trace of antitoxin of the *ovitoxicus* type, but since *B. paludis* antiserum was also employed, there is no occasion to depend on the results with lamb dysentery serum in assessing the nature of the toxic element in the filtrates of that year.

From the neutralisation observations it would seem that the toxic element in all the twelve filtrates investigated in 1931 and 1932 was of the type neutralised by *B. paludis* antiserum, whereas only one of the fifteen fully investigated in 1933 was of this type, the remaining fourteen being of the type neutralised by antisera prepared against *B. ovitoxicus* and *B. welchii* (Wilsdon's type D). It cannot, however, be assumed that the toxic elements were purely of one type or the other, for Glenny, Barr, Jones, Dalling and Ross (1933)[9] point out that the mouse intravenous method of testing does not measure any single toxin-antitoxin reaction, since the test dose of toxin may contain only a few toxic doses of the toxin present in greatest concentration, whereas a larger dose might contain many fatal doses of that toxin and a few fatal doses of another. It can, therefore, only be concluded that the toxic element in the 1931 and 1932 cases, and in Case No. 991 of the 1933 series, was at the least mainly toxin of the type produced by *B. paludis*, whereas that in the remaining cases of the 1933 series it was at the least mainly toxin of the type produced by *B. ovitoxicus*.

It is not contended that these findings are representative, for the cases were not chosen at random. In 1933 there was much selection of material, in that the writers' interests were centred in outbreaks in which the history was atypical, or where serum inoculation had failed to effect control.

The Type of *B. welchii* Isolated from Intestinal Contents.

When the variation in the type of toxic element present in the intestinal contents became evident, the writers determined to obtain cultures of intestinal contents in Robertson bullock heart anaerobic medium wherever possible. Media in test tubes were inoculated with mixed intestinal contents in eight of the sixteen cases detailed above. Pressure of work make it necessary to store these cultures for periods varying from one to three months.

As opportunity arose the isolation of Welch-like bacteria from the original cultures was undertaken. In view of the fact that the nature of the toxic element in these cases was not consistently the same, and was in no case shown to contain the toxin of *B. paludis* as well as the toxin of *B. ovitoxicus*, it was decided to make a number of isolations from each case to cover the possibility of the contents having contained more than one type of *B. welchii*. A freshly boiled tube of

cooked meat medium was heavily seeded from the original culture, and as soon as growth was apparent it was examined by smear preparation for organisms of the *B. welchii* type. In all cases Welch-like forms were at least predominant, and the young subculture was plated out on agar to which about 10 per cent. sterile defibrinated sheep blood had been added. The plates were incubated at 37° C. overnight in a McIntosh and Fildes' anaerobic jar. Several well isolated hæmolytic colonies were picked off and transferred to cooked meat media. The selection of at least six well separated colonies was attempted in each case. These cultures were rapidly passed through two generations in meat broth and then replated. The culture was purified by picking off a single colony, replating after rapid subcultivation, and repeating this process until pure.

Pure cultures which produced a stormy fermentation in milk were tentatively accepted as of *B. welchii*, and were obtained from the original intestinal contents culture in the following cases: Case No. 10 = eleven isolations; Case No. 22B = ten isolations; Case No. 70A = five isolations; Case No. 84 = five isolations; Case No. 85A = four isolations; Case No. 85B = six isolations; Case No. 87 = six isolations; Case No. 90 = nine isolations.

Each of these fifty-six cultures was tested for toxin production by growing the strain in a 250 c.c. bottle of a horse-flesh medium recommended by Dalling. The bottles of media, boiled for twenty minutes and rapidly cooled to about 40° C., were inoculated by discharging 1 c.c. of a six-hour culture of the strain from a pipette at the bottom of the bottle, and were incubated at 37° C. The pH at the time of inoculation was approximately 7.5. Two c.c. quantities of the fluid portion of the resulting culture were withdrawn under sterile precautions for test at the following intervals after inoculations: 18 hours, 6 days, 15 days and 30 days. In view of the small quantity available and the number of examinations necessary, filtration was not attempted, but the samples from the cultures were centrifuged in narrow tubes at 2,500 r.p.m. for half an hour. The supernatant was withdrawn and tested out by intravenous inoculation into mice.

A potent toxin was demonstrated in each of the fifty-six cultures at each examination. At eighteen hours the minimum lethal dose to mice, although not always accurately determined, lay between 0.002 c.c. and 0.0005 c.c. By the sixth day it had fallen to between 0.01 c.c. and 0.005 c.c., a level which was maintained even at the thirtieth day. In mice injected with toxin from eighteen-hour cultures, symptoms developed very quickly and marked excitement and respiratory distress was followed by violent muscular spasms and early death. The injection of toxin from fifteen-day and thirty-day cultures had quite a different effect, the mice remaining apparently normal for quite a period and showing no spasms. They became very dull and frequently showed rotary or circular movements, but death was often delayed until even thirty-six hours after injection. The symptoms following the injection of six-day culture toxin were not constant in character.

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In an attempt to type the toxin present at each time of sampling the antisera, "*B. welchii* G.G., C2748A," "Lamb dysentery serum, Q232," "Lamb dysentery serum, C1852" and "*B. welchii* serum, anti-Wilsdon's type D," were employed. A certain lethal dose, 0.02 c.c. of the eighteen-hour and the six-day cultures, and 0.1 of the fifteen-day and thirty-day cultures, was mixed separately with an equal volume of each antiserum. In the case of the six-day cultures the toxic dose was also mixed with an equal volume of each of the two latter antisera. After incubation at 37° C. for half an hour the mixtures were injected intravenously into mice, which were observed for three days. The results obtained are summarised in Table No. III. The slight variations observed in the toxin produced

TABLE No. III.

NEUTRALISATION OF THE TOXIN PRODUCED BY 56 STRAINS ISOLATED FROM THE EIGHT CASES OF LAMB DYSENTERY.

Toxin produced after Incubation for :	Antitoxic Serum	Protection Afforded
Eighteen hours	*B. welchii*	None in any instance
	Lamb dysentery, Q232	Complete in all instances
	Lamb dysentery, C1852	Complete in all instances
	B. welchii (Wilsdon, type D)	None in any instance
Six days	*B. welchii*	None in any instance
	Lamb dysentery, Q232	Complete in all instances
	Lamb dysentery, C1852	Complete in three instances ; partial in sixteen instances ; none in thirty-seven instances
	B. welchii (Wilsdon, type D)	Complete in thirty-five instances; partial in six instances ; none in fifteen instances
	Lamb dysentery, C1852+*B. welchii* (Wilsdon, type D)	Complete in all instances
Fifteen days and thirty days	*B. welchii*	None in any instance
	Lamb dysentery, Q232	Complete in all instances
	Lamb dysentry, C1852	None in any instance
	B. welchii (Wilsdon, type D)	Complete in all instances

after six days' incubation appear of no significance, for if they are not the result of minor differences in the conditions of growth, but due to inherent properties of the individual strains, they are in parallel with the variations observed in the potency of the toxin produced by accepted strains of *B. welchii*. Furthermore, these minor differences were not confined to strains isolated from one case, but were found to occur indiscriminately, and it can be maintained that the fifty-six strains isolated from the eight cases of lamb dysentery belong to one class of *B. welchii*.

On this evidence it could be contended that this class of *B. welchii* is one characterised by the production of a toxin containing two main fractions : one of the *paludis* type, which is only produced early in the period of cultivation, and which rapidly deteriorates ; and one of the *ovitoxicus* type, produced only after

about six days' incubation, and much more stable. These strains could not be placed with confidence in any one of Wilsdon's groups, for he inferred from the antitoxin he prepared therefrom that his type B, the majority of the strains being isolated from cases of lamb dysentery, produced a small fraction of the *ovitoxicus* type of toxin at even eighteen hours. A more intensive investigation of the nature of the toxin produced by one strain—Case No. 10, isolation 9—was therefore undertaken. The same technique was employed, but examinations were made at more frequent intervals—18 hours, 2 days, 4 days, 6 days and 10 days—and "*B. paludis* antiserum" introduced in place of "*B. welchii* serum." In addition, the protection afforded by combinations of the "Lamb dysentery serum, C1852," or the "*B. paludis* serum" with the "*B. welchii* serum, anti-Wilsdon type D," was observed. The protection which the antitoxins afforded against both a relatively small dose of toxin (5 M.L.D.) and a relatively large dose (100 or 50 M.L.D.) was observed so far as possible in an attempt to overcome, at least in part, the objection to the accuracy of the measure afforded by the mouse intravenous injection of toxin-antitoxin mixtures. The results are set out in Table No. IV. Since there was no difference in the protection afforded by "Lamb dysentery serum, C1852" and "*B. paludis* serum," or the combination of either with "*B. welchii* serum, anti-Wilsdon type D," the results with these sera and combinations are given in one column. These two sera are regarded as identical in their antitoxin content, and are referred to hereafter as of the *paludis* type.

Only the "Lamb dysentery serum, Q232" and a combination of the "*B. welchii* serum, anti-Wilsdon type D," with serum of the *paludis* type protected throughout. Serum of the *paludis* type afforded complete protection at eighteen hours and at two days when the test dose of toxin was small (5 M.L.D.). Since it required the addition of anti-Wilsdon type D serum for the complete neutralisation of a large test dose (100 M.L.D.) of the toxin produced after incubation for these periods, it can be concluded that even early in its growth this strain did produce a toxin containing a fraction of the *ovitoxicus* type. At four and at six days the failure of serum of the *paludis* type to afford protection even when the test dose was small is evidence that the fraction of the *ovitoxicus* type was present in an increased proportion. Since anti-Wilsdon type D serum afforded complete protection after ten days' incubation even with the large test dose, it can be concluded that the fraction of the *paludis* type of toxin had deteriorated entirely, leaving only the *ovitoxicus* fraction. Since this strain was chosen at random, it may be contended that the strains of *B. welchii* isolated from these lamb dysentery cases all belong to the class or group which Wilsdon designed as his type B (the lamb dysentery bacillus).

The writers had received from Dalling a subculture of the original culture of the "lamb-dysentery bacillus" which he isolated in 1923, and which has since been repeatedly subcultured and employed in the production of lamb dysentery serum at the Wellcome Laboratories. The toxins produced by this culture,

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and by cultures of *B. paludis* and *B. welchii* (Wilsdon, type D), also received from Dalling, were investigated in parallel with that of strain 10/9, and the results obtained are set out in Table No. IV. The toxin of the two former organisms was always completely neutralised by the "Lamb dysentery serum, C1852," and the " *B. paludis* antiserum " ; no trace of a fraction of the *ovitoxicus* type being detected. The toxin produced by *B. welchii* (Wilsdon, type D) was always completely neutralised by the homologous antiserum, no fraction of the *paludis* type being present. The "Lamb dysentery serum, Q232" also protected by virtue of its antitoxic content of the *ovitoxicus* type.

There is, therefore, a material difference as regards toxin production between the strains isolated in North Wales and the subculture of the strain which Dalling isolated in 1923, the latter producing a toxin which, as Mason, Dalling and Ross[1] and Mason[12] contended, is identical with that of *B. paludis*.

Discussion and Conclusions.

The writers intended to develop this investigation along lines which would determine whether lamb dysentery was an enterotoxæmia. Certain circumstances, particularly the occasional failure of lamb dysentery serum in the field in 1933, diverted their work into a more general line. Some evidence as to the nature of the disease has, however, been obtained. The detection of a toxic element in each of the intestinal contents filtrates from the twenty-one cases investigated, taken in conjunction with Dalling's observation that the lamb dysentery bacillus could rarely be recovered from sites outside the intestinal canal in cases killed whilst showing symptoms, suggests that the disease is an enterotoxæmia. It might be contended that the site at which toxin is produced is the ulcerated bowel wall, but ulceration is not an essential feature of the disease. It probably results from the necrotic action of toxin produced in the lumen of the gut. Much further work will be necessary before the exact nature of lamb dysentery is determined.

The toxin produced at all periods throughout growth by freshly isolated strains of the lamb dysentery bacillus is neutralised only by the homologous antitoxin. *B. paludis* (Wilsdon, type C) antitoxin fails to neutralise except when the test dose during the earlier period of incubation contains only a small number of M.L.D. ; at later periods it fails entirely. *B. ovitoxicus* (Wilsdon, type D) antitoxin fails to neutralise except when the period of incubation is sufficient to bring about deterioration of the fraction neutralised by *B. paludis* antitoxin. A combination of *B. paludis* and *B. ovitoxicus* antitoxins completely neutralises the toxin at all periods throughout the growth of the lamb dysentery bacillus. It may, therefore, be said that the toxin of the lamb dysentery bacillus contains two chief toxic fractions : one neutralised by *B. paludis* antitoxin, present in high concentration during the early period of incubation (within eighteen hours) and rapidly deteriorating ; the other neutralised by *B. ovitoxicus* antiserum, present in low concentration during the early (up to two days) period of incubation, increasing as incubation is prolonged and relatively stable.

TABLE No. IV.

NEUTRALISATION OF THE TOXINS OF THE LAMB DYSENTERY BACILLUS (RECENTLY ISOLATED STRAIN, 10/9); THE LAMB DYSENTERY BACILLUS (LONG ISOLATED STRAIN); *B. paludis* AND *B. welchii* (WILSDON, TYPE D), BY STANDARD ANTITOXINS.

Period of incubation	Dose of toxin (M.L.D.S.)	Toxin of: L.D. bacillus (strain 10/9) + equal volume of:				L.D. bacillus (long isolated) + equal volume of:			*B. paludis* + equal volume of:			*B. welchii* (Wilsdon, type D) + equal volume of:		
		L.D.	P.	D.	P+D	L.D.	P.	D.	L.D.	P.	D.	L.D.	P.	D.
18 hours	5	LL	LL	††	LL	LL	LL	††	LL	LL	††	LL	††	LL
	100	LL	††	††	LL	LL	LL	††	LL	LL	††	Culture toxic only in large doses		
2 days	5	LL	LL	††	LL	LL	LL	††	LL	LL	††	LL	††	LL
	50	LL	††	††	LL	LL	LL	††	LL	LL	††	LL	††	LL
4 days	5	LL	††	L†	LL	LL	LL	††	Culture toxic only in very large doses			LL	††	LL
	50	LL	††	††	LL	LL	LL	††				LL	††	LL
6 days	5	LL	††	L†	LL	Culture toxic only in very large doses			No toxin demonstrable			LL	††	LL
	50	LL	††	††	LL							LL	††	LL
8 days	5	LL	††	LL	LL	No toxin demonstrable			...	...	...	LL	††	LL
	50	LL	††	LL	LL				...	...	...	LL	††	LL
10 days	5	LL	††	LL	LL	...	...	...	...	...	...	LL	††	LL
	50	LL	††	LL	LL	...	...	...	...	...	...	LL	††	LL

L.D. = Lamb dysentery serum, Q232. P. = *B. paludis* antiserum and lamb dysentery serum, C1852.
D. = *B. welchii* serum, anti-Wilsdon's Type D. L = Lived. † = Died.

A strain of the long isolated and much subcultivated strain of the lamb dysentery bacillus used at the Wellcome Physiological Research Laboratories for the production of lamb dysentery serum produced no fraction of the *ovitoxicus* type at any period in its growth. Dalling has, however, shown that a subculture of the original isolation does produce a toxin similar to that of the writers' recently isolated strains. Judging from the nature of the serum produced at the Laboratories, it would seem that this change occurred prior to the production of the serum issued for field use in 1933, and explains the lack of antitoxin of the *ovitoxicus* type in that serum.

It is appreciated that the methods employed in the investigation of the nature of the toxic element in the intestinal contents of these cases were not capable of demonstrating the absence of one fraction of the toxin of the lamb dysentery bacillus, in the presence of another. Yet, in view of the fact that the test doses probably contained several M.L.D., it is somewhat surprising that in no case was a mixture of the toxic fractions demonstrable. Since one or other of these fractions may at the least be predominantly present in the intestinal contents, it is necessary that lamb dysentery serum for field prophylaxis contain a high content of antitoxin of both types. The effect of the absence of antitoxin capable of neutralising toxin of the *ovitoxicus* type is illustrated by the failure of 1933 serum to effect a satisfactory degree of control on certain farms in North Wales.

The variation in the type of toxin present in the intestinal contents of these cases might suggest that either of two organisms of the *B. welchii* group may cause lamb dysentery. With a view to covering this possibility, multiple isolations were made from lamb dysentery cases. Since each of the fifty-six strains isolated from eight cases conformed to one type, it can be conluded that this type of *B. welchii*, the lamb dysentery bacillus (Wilsdon, type B), alone is responsible for the disease.

Acknowledgment.

The writers desire to acknowledge their indebtedness to Mr. T. Dalling, M.R.C.V.S., for much helpful advice and criticism throughout this investigation.

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NOTE.—BY T. DALLING, M.R.C.V.S., *Wellcome Physiological Research Laboratories, Langley Court, Beckenham, Kent.*

Montgomerie and Rowlands have referred to the strain of micro-organism isolated by us in 1923 from a case of lamb dysentery. This strain was used by us for some years in the production of toxin and antigens with which horses have been injected for the production of hyperimmune serum. It was not until we became acquainted with the fact, through work carried out in conjunction with these authors, that the *ovitoxicus* type of toxin could be isolated from the intestinal contents of lambs dead of lamb dysentery that we examined the culture for its ability to produce this toxin. To our astonishment we found that the cultures in use in 1932 (which were subcultures, many generations old, of the original 1923 culture) failed to produce any detectable *ovitoxicus* type of toxin in fair quantity. It is evident that at some time between 1923 and 1932 this strain had lost its power to produce *ovitoxicus* toxin in amounts detected by the methods we employ. This is further borne out by the fact that an examination of the sera prepared at various periods showed that the older samples, produced prior to 1932 (of which Q.232 was one), contained *ovitoxicus* antitoxin, while more recently prepared samples did not contain such antitoxin as far as our tests could show. The question of this loss of power of a strain of *B. welchii* to produce certain types of toxin is being investigated.

ELECTRICAL STUNNING OF ANIMALS.

By ANTON WEINBERGER.

Munich.

PROFESSOR J. ROOS and Mr. S. Koopman, of Utrecht, have published in THE VETERINARY JOURNAL of June, 1934, their studies of electrical stunning, which, however, cannot be allowed to be passed over without being contradicted, as they give an utterly wrong idea of the electrical stunning of animals to be slaughtered, for this method is being used to-day at over one thousand places with great success, and the electrical stunning is based on the opinion of many hundreds of renowned veterinary surgeons.

Among the authorities who have gone to the trouble of studying thoroughly electrical stunning in Germany, I wish to name the well-known physiologist at the Veterinary College of Leipzig University, Professor Dr. Gildemeister; also

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Mr. Alvensleben, chief engineer at Berlin, president of the Professional Union for Electrotechnics, a recognised authority in the field of accidents cause by electricity; Professor Dr. med. W. Schulze, of Berlin, who is investigating electrical stunning and its use for human beings; University Professor Dr. Max Muller, of Munich, who since the year 1927 has been continuously engaged in the electrical stunning of animals to be slaughtered, and at whose suggestion the method of electrical stunning was worked out by me.

After having made all fundamental tests, and also since I have carried out electrical stunning myself with several thousands of cattle, I think I may consider myself entitled to give an opinion on the electrical stunning of animals for slaughter, for these are the animals concerned.

In the first instance it may be mentioned that with swine, sheep and calves, electrical stunning does not work faultlessly, when *alternating current* is used, unless at least 500/700 milliamperes with 70 volts pass through the brain or through the prolonged spinal marrow for ten to fifteen seconds. In carrying out the electrical stunning with these animals, they are grasped behind the ears or at the temples with stunning tongs with insulated handles. The electrodes of these tongs are supplied with sponge insertions dipped previously into salt water of high percentage (at least 20 per cent.) to diminish the resistance of transition of the skin. In a fraction of a second the animals are unconscious, and one never hears a cry, nor are there any defensive movements to be noticed. Then the degree of unconsciousness increases gradually, similar to the inhalation narcosis, and when ten to fifteen seconds later the tongs are taken off the animal the deep unconsciousness continues for three to five minutes, during which time the animal must be bled. At the moment of cutting in there is a strong electrospasm, which disappears, however, immediately as soon as the tongs are taken off, and the muscles of the animal are completely relaxed.

Animals are stunned in the same manner when interrupted direct current is used. The tension is always between 60 and 70 volts.

The stunning of animals for slaughtering such as is carried through everywhere represents an over-stunning, that is, the dose of the current is so large that it would kill the animal if allowed to act for a prolonged time, that is, after a few minutes the breathing and action of the heart would stop.

The current produces in the brain a very strong anæmia, which is proved best with the stunning of poultry, which immediately get a lighter comb which recovers its red colour after the birds have regained consciousness if they are not killed.

The electrical stunning is a method of stunning to which no objection can be made, and it would have been an easy matter to Professor Dr. Roos as well as to Mr. S. Koopman to control the experiments at any slaughter-house where electrical stunning has been in use for some time. I may be allowed to mention here expressly that, knowing Professor Dr. Roos's attitude from the beginning, I offered to place all testing apparatus at his disposal free of charge so as to enable him to determine border-values. He declined this offer however.

The electrical stunning of animals to be slaughtered has nothing whatever in common with an electric permanent narcosis, because an electric permanent narcosis cannot be effected except with interrupted direct current, and but a few milliamperes and tensions of 8–10 volts are needed for it. With rabbits even 4–5 volts are sufficient. Professor Leduc, of Nantes, carried out these experiments as long as thirty years ago with absolute success; and he also effected local stunnings with interrupted direct current, which is likewise easy to imitate.

The object of electrical stunning for slaughtering purposes is by no means to produce a slight narcosis, but an extremely rapid and very strong stunning is to be effected thereby, which lasts a few minutes, so as to render the bleeding possible in the unconscious state.

Permanent narcosis with a few milliamperes and very low tension are used for operations or when bleeding horses and cattle for the purpose of obtaining serum. Only interrupted direct current can be used for this purpose, and the animals may be kept under tension for any length of time. Though an anæmia of the brain occurs, too, with interrupted direct current, the electrolytical phenomena also manifest themselves. An interesting experiment to observe also is the following electrolytical phenomena: A trout or a carp is stunned with small tongs of a few volts (about 20–30). If ordinary alternating current of the same tension is used, the fish is stunned too much (over-stunning), it is true, but it does not alter its appearance in any way. If interrupted direct current is used, however, the fish changes its colour at those places where the electrodes touch, just as if the fish had been boiled blue in salt water, which indicates that the electrolytic effect is noticeable at the mucous membranes of the fish head.

I was horrified to see that before making the electrical stunning experiments Professor R. Roos and Mr. Koopman narcotised the animals by chemical means. The entire effect of the electrical stunning was, of course, destroyed thereby, and only signs of irritation could be noticed. Whoever makes experiments in such a manner had better leave it alone, for he proves that he is not trying seriously to obtain limits.

Dogs and cats are the worst animals to be experimented upon for stunning purposes. With a dog the passage of current with a tension of 60–70 volts would amount to at least 700–1,000 milliamperes if the tongs are dipped into 20 per cent. salt water, as is done with normal stunning at the slaughter house, for the dog's head is proportionately small, as is also the resistance.

The total result of the experiments of Professor Dr. Roos and Mr. S. Koopman is an excellent example of how the experiments should not be made, and that from such experiments one must not draw any conclusions regarding the stunning of cattle to be killed; for either the experiments are carried out in such a manner that at least 500–700 milliamperes pass through the animal, or one must limit oneself to experiments of permanent stunning with very low intensities of current. Dr. J. Kok and Dr. A. van Harreveld, of the Physiological Institute of Utrecht,

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have come to quite a different result, which has been published in the journal *Tijdschrift voor Diergeneeskunde*, Ed. No. 2, of January 15th, 1934.

Such assertions only produce uneasiness among the public, and it is interesting that those who have carried out the experiments incorrectly, and who have no experience at all in the field of cattle stunning, consider themselves competent to draw unfavourable conclusions from electrical stunning. In Germany only one manager of a slaughter-house has objected to electrical stunning, but he has been disavowed by all his colleagues.

For the rest, Professor Dr. Roos is said to have worked out an opinion for the rabbits in Germany which differs very considerably from his former ones. In any case, all managers of slaughter-houses and manufacturers of tinned meat who have an electrical stunning plant will confirm my statements in every respect, for at present more than 1,000 electrical stunning plants are working, and the opinion of about 1,000 veterinary surgeons will no doubt be more convincing than the experimental results of a scientist who has started from an entirely wrong premise, as proved by his experiments. In England, as well as in Denmark and partly also in Poland, all pigs, and in Belgium sheep also, have been bled without any stunning at all up to a short time ago, whilst after the introduction of electrical stunning these animals have the advantage of the humane killing method. Any doubters of the electrical stunning should visit slaughter-houses where an electrical stunning plant has been installed, and they will at once realise the humane slaughtering method, and they will gain the conviction that the experiments made by Professor J. Roos with dogs and cats have been carried out incorrectly.

At present at least 50–60 million pigs and about 10 million sheep are stunned electrically every year, and the opinion of the veterinary managers of those slaughter-houses is excellent. It is true the electrical stunning method is heavily attacked by the makers of the bolt-shooting apparatus, which can readily be understood, as more than 100 pigs can be stunned electrically for the price of one single cartridge.

PROSPECTS OF VETERINARY SCIENCE IN AUSTRALIA.

By R. T. PURSELL, B.V.Sc. (University of Sydney).
New South Wales.

BEING a graduate from the Sydney University here on a world tour, my impressions of veterinary science in England, compared with the position and prospects of the profession in my own country, might be of interest.

The course in Australia is four academic years, but there is a strong likelihood of it being increased to five in the near future. I graduated in December, 1933, and arrived here in May, 1934. I have completed a tour of Europe, taking in part of France, Switzerland, Germany and Belgium ; then on my return had a hurried round trip through England, Scotland and Wales, and am now about to leave for

the Congress being held in America. The impression I have gained by interviewing members of the profession in different parts of the country, is that graduates in veterinary science, for a number of years, will be rapidly absorbed into quite reasonably paid positions, or else will enter practice ; the same, up to a point, might be said of Australia. There we have only one school of veterinary science, situated at Sydney ; the other, at Melbourne, having been discontinued some years ago.

Perhaps a brief comparison between the United Kingdom and Australia might be made at this juncture. Australia, with its 2,954,581 square miles, is almost equivalent to 25 times the size of Great Britain and Ireland. As regards livestock, its horses numbered in 1931, 1,776,000 (United Kingdom, 1,194,000) ; its cattle, 12,661,000 (United Kingdom, 7,955,000) ; its sheep, 110,619,000 (United Kingdom 26,374,000).

As you have here, so we have a considerable amount of administrative veterinary work being carried out, chiefly on the side of disease control, necessitating a large staff of veterinarians, and so opening up the following avenues for the graduate.

In the Government Service we have the Stock Branch of the Department of Agriculture ; here the work of the veterinary surgeon covers both administrative and field work. The administrative work includes the administration of a number of Acts relating to stock, such as the Quarantine Act, and different Acts dealing with disease in livestock. The field work includes dairy herd inspection, clinical examination for mastitis, T.B. testing, etc.

Then there is the work connected with the meat industry. Here there are, at the present time, limited appointments to the Commonwealth Meat Inspection and State Meat Inspection services. The former is concerned with meat for export, and the latter chiefly with meat for local consumption. The type of work is, of course, principally ante- and post-mortem examination of cattle and sheep.

In the metropolitan area of Sydney the slaughter of livestock for food purposes is centralised at the Homebush Abattoirs and several meat-canning factories. In the country we have no properly organised system of killing, the slaughtering being carried out by numerous small butchers scattered all over the country. This naturally brings with it the attendant disadvantages, such as inadequate inspection of meat, this duty often being performed by a member of the local constabulary with a rudimentary knowledge of meat inspection, or by the stock inspector, whose work in connection with disease control over large areas leaves him insufficient time to give to the actual meat inspection. Added to the above is the fact that a small town may have as many as five or six separate slaughter-houses at considerable distances apart, and these towns might be twenty to forty miles distant from each other. So that eventually, I think, we will have country killing centralised in the larger towns, with the appropriate veterinary supervision.

Next we may consider the research side of the profession. In New South Wales there are a number of research stations. Close to Sydney is the Glenfield

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Research Station, perhaps the largest, handling different problems such as black disease in sheep, and carrying out routine work such as agglutination tests for contagious abortion, biological tests for T.B., etc.

Other research stations are situated at Sydney, Canberra (the Federal capital), Trangie, and a number in the other States. The men working at the stations are paid either by the Government, by various research scholarships, or by the Council for Scientific and Industrial Research.

The salaries paid by the New South Wales Government are, at the present time, subject to heavy cuts, and are, on the whole, lower than those of Queensland and Victoria.

Stock Inspectors.—Pastoral and agricultural areas are divided up into a number of districts, and for each district there is what is known as a Pastoral Protection Board, elected from stock and land owners. The P.P.B. pays the salary of a man appointed by the chief veterinary officer of the State, this man being a stock-inspector. His work lies chiefly in the control of outbreaks of disease and movements of stock in and out of his area, in the maintenance of stock routes, the control of the rabbit pest and dairy inspection. In the past the stock inspector has been a stockman with a limited knowledge of disease, and now, throughout the country, is being replaced by the qualified man. The salary of a stock inspector varies according to the district which he controls. In the wealthy districts towards the eastern part of New South Wales the range is something like £400–£600 with expenses; in the western plain country, with its large areas of poorer country, the salary tends to be less, say, £300–£500.

Private Practice.—This at present tends to be limited to the closer settled areas and large towns. We have a large number of registered veterinary surgeons, *i.e.*, men who prior to the Veterinary Surgeons Act, N.S.W., had practised as veterinary surgeons, and a limited number of qualified men, these being centred in our larger cities, *viz.*, Sydney and Melbourne, or in the coastal districts.

I do not think that at the present time country practice offers a very attractive future to the graduate because of the question of long distances between properties and towns—being anything from 20 up to 100 miles in the less settled areas—and because of the generally larger herds and flocks, with the subsequent lower value per head of stock than in England. The grazier is usually content to carry out his own treatment, even if it does mean the loss, perhaps, of say fifty or more sheep, where the assistance of a veterinarian might, perhaps, materially reduce this figure.

In conclusion, the growth of the Faculty of Veterinary Science in Sydney has been very rapid in the last few years, there being three times the number of students there were four years ago: the number of students now being something like 110.

The work is there, and this strengthening of the veterinary profession should materially aid the progress and prosperity of our livestock industry.

MASTITIS IN CATTLE.

By J. F. D. TUTT, F.R.C.V.S.

Winchester.

THERE is probably no other disease encountered in general practice which is attended with less satisfactory results to owner and practitioner than the varying forms of mastitis. It is a regrettable fact that up to the present there is no known remedy which can be relied upon, not only to save the affected animal, but, more important still, to restore the affected quarter to its former functioning power.

During the past six months a fairly extensive trial has been given to Seelegmann's method of infusing a solution of entozon into the affected quarter or quarters, and it can be said that, providing the affected quarter has not become markedly indurated and that the condition is purely of a streptococcal nature (I exclude the virulent streptococcus pyogenes infection, which in most cases is attended with a rapidly fatal termination), the results are distinctly encouraging. The distributors (Bayer Products, Ltd.) point out that the entozon should not be injected, but infused. The procedure is very simple, and the intravenous outfit supplied by Messrs. Arnold & Sons answers admirably for the purpose, the needle being replaced by an ordinary milk tube. In the absence of a suitable container, the bottles in which the improved calcium gluconate solution are now sent out can be utilised, and the same procedure is then adopted as for intravenous injection. The concentration of entozon advised is 1.1250, and it should be infused at body temperature (37–40° C., or 99–104° F.). It will be found that the solution will gravitate into the quarter as rapidly and readily as into a vein. Before infusing, the quarter should be stripped as dry as possible. After infusing, massage is adopted for about ten minutes, and then the quarter is again stripped out. As a rule, it will be found that all the solution can be recovered with the exception of about two ounces. The treatment is repeated in three days. Bacteriological examination of milk samples is advisable at intervals of ten to fourteen days to determine the headway (in the absence of induration) of treatment. If possible, $1\frac{3}{4}$ litres should be infused into each quarter.

In cases of marked induration, or where the secretion is of the bloody, highly offensive type, indicating the presence of putrefactive bacilli (such as *B. necrosis*, etc.), better results follow the injection into the actual udder tissue of parenchymatol-silver-albumen (parenchymatol-silber-eiweiss of Kommer & Co., London). For this purpose a long needle such as a lumbar puncture needle is employed. The needle is thrust as deeply as possible into the udder tissue of the affected quarter, and five injections of 20 c.c. each made at five different points. The injected quarter is massaged and milked out in twenty-four hours. The treatment is repeated in seventy-two hours.

Medicinal remedies per os cannot be recommended save as a possible placebo to the client, or as an adjunct in the nature of a febrifuge. Some few years ago

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formalin, in one-dram doses in two pints of cold water morning and night, was recommended as a specific. The chief effect in a large number of cases observed was diarrhœa, which in not a few cases was blood stained, and it has long been discarded.

Concluding with the value of vaccines. The stock variety can only be regarded as of the " hit or miss " type, and the best results follow the use of autogenous vaccines. Where several cases of mastitis occur in a herd, it will often be found that a particular species of streptococcus is responsible, and it is a good plan to have an autogenous vaccine prepared which is injected not only into those already affected, but into cows due to calve. In this latter case, three injections at intervals of five to six days are given before parturition, and a similar number at the same interval after parturition. It is apt to be lost sight of that the so-called " kernels," " warts," " spiders " met with in the teat canal owe their origin to a streptococcus infection, and that a similar vaccine in a susceptible herd will reduce this tendency. It should be emphasised that the udder of the cow is the most vulnerable part of her anatomy, and that the utmost care and cleanliness are essential not only during the period of lactation, but in the drying off process. Guernseys and Jerseys cannot be " dried off " in the same way as Shorthorns.

The great advantage, too, of non-interference of the udder in cases of milk fever (now rendered possible by the intravenous injection of calcium gluconate, with no risk of sloughing or abscess formation, as in the case of subcutaneous injection, denied by pundits, but confirmed by herdsmen all over the country and responsible for the opposition to this treatment in the best pedigree herds) is that not a few of the organisms responsible for mastitis are not only aerobic, but are present in the vast majority of cows' udders, often in a more or less inactive state. The inflation of the udder in such cases with either oxygen or air will often bring them into activity and set up a mastitis, usually ascribed in the past to undue attention to the cleanliness of the teats and milk tube before inflation.

FOWL TYPHOID IN KENYA COLONY*

By J. R. HUDSON, B.Sc. Lond., M.R.C.V.S.,
Division of Veterinary Research, Kabete, Kenya.

Introduction.

THE presence of a " disease of fowls due to a bacillus of the dysentery group " in Kenya was first reported by Montgomery in 1912. Owing to the heavy mortality that occurred at that time in a flock of fowls at Kikuyu, the disease became known as " Kikuyu fowl disease." This name is still in common use in the Colony. Further references to the disease are to be found in the annual reports of the veterinary pathologist, Nairobi, and later in the reports of the Chief Veterinary Research Officer. In 1922, Kearney published an account of some experi-

* Received March 24th, 1934.—ED.

ments, in the course of which he ascertained that the usual method of infection was by the ingestion of bacilli excreted in the fæces of diseased birds. He showed that domesticated ducks and pigeons were immune, and he noted the low susceptibility of fowls imported from South Africa when compared with the high susceptibility of the local native birds. It was not until thirteen years after its discovery, however, that the identity of Kikuyu fowl disease with fowl typhoid was realised.

TABLE I.—SUMMARY OF POST-MORTEM EXAMINATIONS MADE ON POULTRY AT KABETE LABORATORY, FROM JUNE 1st, 1931, to MAY 31st, 1932.

	Fowl Typhoid	Helminthiasis	Fowl-pox and Roup	Peritonitis	Visceral Gout	Coccidiosis	Tuberculosis	Neurolymphomatosis	Blackhead	Pullorum Disease	Various	Cause of death not determined; usually owing to decomposition.	Total of each group examined
Adult fowls... ...	38	11	14	6	4	—	—	1*	—	—	21	27	122
Chicks	—	5	4	—	—	3	1	—	—	3	3	9	28
Turkeys	1	—	2	—	1	—	—	—	2	—	—	4	10
Turkey chicks ...	—	—	—	—	—	1	—	—	—	—	1	4	6
Goslings	—	—	—	—	—	—	—	—	—	—	—	3	3
Ducks	—	—	—	2	—	—	—	—	—	—	—	6	8
Totals	39	16	20	8	5	4	1	1	2	3	25	53	177

* An imported White Leghorn cock.

During recent years the economic importance of pullorum disease in Europe and America has overshadowed fowl typhoid and, indeed, most other poultry diseases. It is probably on this account that text-book descriptions of fowl typhoid, especially from the point of view of immunity and carriers, are so unsatisfactory. In Kenya pullorum disease, as it is known in these other countries, that is, as a serious disease causing a heavy mortality in incubator hatched chicks, has only recently been diagnosed for the first time.

Table I shows the importance of fowl typhoid to the poultry farmer in Kenya to-day. This table may be compared with a similar table for the same period printed in the Annual Report of the Poultry Research Laboratory at Goring, England, for 1931–1932. At Goring only fourteen cases of fowl typhoid were encountered during the post-mortem examination of 2,131 birds.

There is a tendency, particularly among Continental workers, to consider fowl typhoid and pullorum disease as different manifestations of one condition. The writer believes that this theory is not yet justified, and holds that the epizöotiology of the two diseases in Kenya is against it. For a number of years outbreaks of fowl typhoid have occurred in the flocks of most of the larger breeders.

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Incubators are now in use in certain of these flocks, and yet the only outbreak of pullorum disease that has occurred in incubator-hatched chicks was associated with carrier hens imported from England. In two outbreaks on another farm, where two and three fourteen-day-old chicks respectively died out of two hen-hatched sittings, the eggs were from imported birds.

Occurrence.

Fowl typhoid occurs throughout the Highlands of Kenya. A study of the cases received at Kabete over a number of years shows that there is no marked seasonal incidence, nor does the annual rainfall appear to affect the annual incidence. The disease is usually seen in fowls and turkeys. Montgomery first reported, cases in guinea-fowl in 1914–1915. In spite of the large number of outbreaks that occur annually, the disease has never been encountered naturally in young chicks. No cases of mortality in chicks during outbreaks in older birds have been reported, and in the course of many bacteriological examinations of young chicks sent to the laboratory for diagnosis the writer has never isolated the organism of fowl typhoid.

Walker, in 1922, reported that young birds aged from one week to six months were fully susceptible, both on feeding and on inoculation. The writer's experience is that chicks are very easily infected experimentally. Thus a culture of the organism inoculated to three chicks, three days old, caused the death of two, two and three days later. In another experiment, three chicks one week old died four, four and five days respectively after inoculation. From the liver of all the dead birds the organism was recovered in pure culture. In a third experiment three chicks were fed a few drops of an emulsion of a recently-passaged strain by means of a pipette passed into the œsophagus. The first chick died the following day from an undetermined cause. The other two died four days after feeding, and the organism was again recovered in pure culture from the liver.

Beach and Davis (1927) reported a case of heavy mortality in incubator-hatched chicks due to fowl typhoid. Beaudette, in his report to the Eleventh International Veterinary Congress, stated that about one-third of the outbreaks of fowl typhoid found in New Jersey had occurred in young chicks. European writers, however, generally regard the disease as essentially one of adult stock. Considering the prevalence of the disease, it is difficult to account for the complete absence of cases in chicks in Kenya. An outbreak which, unfortunately, was not confirmed bacteriologically, was reported in March, 1932, in a flock of pullets two months old. This suspected outbreak occurred several months after a severe outbreak, confirmed bacteriologically, in adult stock on the same farm. The pullets were incubator-hatched and had been reared on newly-enclosed ground not previously used for poultry.

The origin of a new outbreak may in certain cases be traced to the introduction of an infected bird ; but is more often obscure. It is frequently stated that native birds are a source of infection, and a warning to this effect is given in the Report

of the Department of Veterinary Science and Animal Husbandry, Tanganyika Territory, for 1928. While it is, of course, possible to introduce a carrier native fowl, in view of the high susceptibility of native birds generally and the fact that the outbreak usually starts with the death of the native fowl, another explanation may be more probable, that the carrier bird was already present in the run. Owing to its high susceptibility the native bird becomes infected and dies. The consequent increased infectivity of the ground and the increased virulence of the recently passaged organisms may then be sufficient to break down the partial immunity of some of the other birds.

TABLE II.

Bird No.	Test on 17.3.32 1/25 1/50 1/100	Test on 4.4.32 1/25 1/50 1/100	Test on 25.4.32 1/25 1/50 1/100	Test on 30.6.32 1/25 1/50 1/100	History
1054	+ + + / + + +	+ + + / + + +	—	—	Died 4.5.32. P.-m. not characteristic ; cultures from liver and ovary positive
1085	— — —	— — —	+ + / + + +	— — —	Died 3.7.32. P.-m., fowl typhoid ; cultures from liver and ovary positive

That outbreaks can originate from breakdown of the immunity of carrier birds is shown by the histories of such birds as the two Rhode Island Red hens, 1054 and 1085 (Table II). These birds were survivors of a severe outbreak which lasted from December, 1931, to the end of January, 1932. The whole of this flock of pure-bred birds, which will be referred to again, was taken over temporarily by the laboratory on March 17th, 1932, for an experiment to determine if outbreaks could be controlled by testing and segregating the clean birds, the methods used to control pullorum disease. Bird 1054 was a heavy reactor at the first test and was placed in a run with other reactors. She died on May 4th. Bird 1085 gave no agglutination in the 1 : 25 tube at the first test, and was placed in a run with other non-reactors. On April 25th, her serum agglutinated strongly and she was removed from the clean run. On June 30th, her serum again gave no agglutination, even at 1 : 25, but four days later she died. Both 1054 and 1085 showed lesions typical of fowl typhoid on post-mortem examination, and from the liver and ovary of each *B. gallinarum* was isolated in pure culture.

The factors responsible for these relapses are not fully known. The histories of various outbreaks reported to the writer show that although no new birds are introduced, the removal of a flock of birds to clean ground on a new farm may precipitate an outbreak. Also on several occasions, when there has been no justification for assuming the introduction of fresh infection, the strain imposed on a hen when sitting has led to death and the start of an outbreak.

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Symptoms and Pathology.

The symptoms and pathology of the disease in Kenya differ in no way from those observed in other parts of the world. The acute form of the disease is most frequent, death occurring within twenty-four hours of the first signs of illness. In the writer's experimental cases death has occurred from four to twenty days after the feeding of virulent cultures. Following the inoculation of virulent cultures death may occur in as short a period as two days. Sick birds show diminished appetite and great thirst. They stand with wings drooping, eyes closed and feathers ruffled. The comb and wattles are often cyanotic, but may be pale. Regurgitation of slimy green ingesta is sometimes seen. Diarrhœa is usually observed, the fæces being sometimes dark-green and slimy, sometimes yellow and chalky. The temperature, elevated at first, falls rapidly before death. In cases that survive recovery is slow, the birds often taking a month to become normal. During this time the feathers are rough and the birds have a listless and untidy appearance.

At autopsy the liver is somewhat enlarged and, though usually mottled with fine pale spots and hæmorrhages, may be diffusely pale or bronzed. Stress is often laid by text-books on the great enlargement of the liver. In the writer's experience birds which die with greatly enlarged livers are frequently negative bacteriologically, and many are undoubtedly cases of leukæmia. The spleen is constantly much enlarged, being often three or four times its normal weight. Usually it is dark, somewhat blue in colour, and soft in consistence. The intestines show catarrh and swelling of the mucosa from the pylorus to the junction of the cæca. Fine hæmorrhages are often seen, being, when present, most marked in the duodenum. The lungs may be normal or congested. The heart is usually normal, but fine hæmorrhages on the epicardium may be observed. The oviduct is usually congested and often shows fine hæmorrhages in the mucosa. The mouth and pharynx are almost always cyanotic and contain dirty, stringy mucus.

Diagnosis.

A definite diagnosis cannot be made on either the symptoms or the lesions observed post-mortem. During the course of routine examinations of birds received at Kabete it is usual to suspect fowl typhoid whenever a dead bird that shows well-developed breast muscles and an abundance of abdominal fat is sent for diagnosis. As a matter of routine, cultures on agar are made from the liver of all birds that are received, and a report is not given until the results of this examination are available. As fowl cholera has never been diagnosed in Kenya, the appearance of the twenty-four hour agar culture is usually sufficient for diagnosis. When the history makes the diagnosis doubtful or the bird is known to have been vaccinated previously, carbohydrate fermentation tests are made. Even if decomposition is moderately advanced, no difficulty as a rule is experienced in isolating the organism in pure culture from the liver. When an unpreserved

TABLE III.—CARBOHYDRATE REACTIONS OF *B. Gallinarum* WITH SOME OTHER RELATED BACTERIA ENCOUNTERED IN KENYA.

	AVIAN										BOVINE					CANINE	
	Pathogenic										Non-pathogenic	Pathogenic				Non-pathogenic	Non-pathogenic
	B. gallinarum								*B. pullorum*		Fæces of fowl	Kenya calf para-typhoid	Kenya calf enteritidis	Kenya calf proteus	Liver of calf	Fæces of dog.	
Strains	G7, 12, 15, 19	G5	G1, 6, 11, 22	G2, 4, 13, 16, 17, 18, 21, 23	G10, 24	G3, 8	G14	P28	P29	27	Para. 1, 2, 3, 4, 5, 6, 7	Ent. B.	Pr. 1	S1	S2		
Days	1 3 7	1 3 7	1 3 7	1 3 7	1 3 7	1 3 7	1 3 7	1 6 14	1 3 20	1 3	1 14	1 14	1 14	1 3	1 3		
Dextrose	A A A	A A A	A A A	A A A	A A A	A A A	A A A	A A A	A A A	AG AG	AG AG	AG AG	AG AG	AG AG	AG AG		
Lactose	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— —	— —	— —	— —	— —	— —		
Saccharose	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— —	— —	AG AG	— —	— —	— —		
Mannite	A A A	A A A	A A A	A A A	A A A	A A A	A A A	A A A	A A A	AG AG	AG AG	AG AG	— —	AG AG	AG AG		
Dulcite	A A A	A A A	A A A	A A A	A A A	A A A	A A A	— — —	— — —	ag ag	AG AG	AG AG	— —	— —	— —		
Maltose	A A A	A A A	A A A	A A A	A A A	A A A	A A A	*a — —	*a a —	AG AG	AG AG	AG AG	A AG	AG AG	AG AG		
Arabinose	A A A	A A A	A A A	A A A	A A A	A A A	A A A	A A A	A A A	AG AG	AG AG	AG AG	— —	A A	AG AG		
Dextrin	A A A	A A A	A A A	A A A	A A A	A A A	Not tested	— — —	— — —	— —	Not tested	Not tested	Not tested	— —	— —		
Filtered Dextrin	a a a	a a a	a a a	a a a	a a a	a a a	a a a	Not tested	Not tested	Not tested	— —	— —	— —	Not tested	Not tested		
Raffinose	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— —	— —	— —	— —	— —	— —		
Rhamnose	a A A	a a A	— A A	— a A	— — A	— a A	— — A	— — —	— — A	AG AG	AG AG	AG AG	— —	A A	AG AG		
Salicin	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	AG AG	— —	— —	AG AG	— —	— —		
Sorbite	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— a A	— a A	AG AG	AG AG	†AG AG	— —	— —	— —		
Xylose	a A A	a A A	a A A	a A A	a A A	— a A	— a A	— — —	— — —	AG AG	— —	AG AG	A A	AG AG	AG AG		
Inosite	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— — —	— —	— —	— —	— —	— —	— —		
Inulin	...	— — —	...	...	...	...	...	— — —	...	...	— —	— —	...	...	...		
Adonite	...	— — —	...	...	...	...	...	— — —	...	...	— —	— —	...	...	...		
Erythrite	...	— — —	...	...	...	...	...	— — —	...	...	— —	— —	...	...	...		
Galactose	...	A A A	...	...	...	...	...	A A A	...	...	AG AG	AG AG	...	...	...		
Nutrose	...	— — —	...	...	...	...	...	— — —	...	...	— —	— —	...	...	...		
Glycerine	...	— — A	...	...	...	...	...	— — —	...	...	— —	— —	...	...	...		
Levulose	...	A A A	...	...	...	...	...	a A A	...	...	AG AG	AG AG	...	...	...		
Days of incubation	...	1 4 20	...	...	...	...	...	1 4 20	...	...	1 4 20	1 4 20	...	1 14	1 14		
Litmus milk	...	— — Alk	...	...	...	...	...	— a Alk	...	...	— — Alk	— — a	...	— —	— —		
Hetsch (mannite)	...	AO AO AO	...	...	...	...	...	U U U	...	...	AO AT AC	AO AT AC	U U	AO AC	AO AC		
Indol production	...	—	...	...	...	...	...	—	...	...	—	†—	+	+	—		

A = strong acidity; a = weak acidity; G = marked gas production; g = weak; Hetsch U = unchanged; AO = acidity only; AT = acid and turbidity; AC = acid and clot.
For Hetsch medium, see *Zentral. fur Bact. Orig.*, xxxiv, p. 580.

* A trace of acidity in the inverted collecting tube only.

† Most strains of *B. enteritidis* from calves in Kenya do not ferment sorbite, and some produce indol.

liver is received, having been several days in the post however, plate cultures on McConkey's bile-salt neutral red medium are used.

In order to give an earlier diagnosis, the author has tried the rapid whole-blood agglutination test when birds have been received alive. Although in a few cases a positive result has been obtained, it would seem that the majority of birds die before the development of agglutinins in their blood. The examination of a blood smear stained with Giemsa has proved of greater value. Polymorphonuclear leucocytes are greatly increased in numbers in the blood of fowls dying of fowl typhoid. Owing to the rarity of other diseases in which large numbers of polymorphonuclear leucocytes occur in the blood, their presence justifies a diagnosis of " very probably fowl typhoid."

Ætiology.

Judging by the relatively great susceptibility of native fowls as compared with imported birds, a fact first reported by Kearney (1922), the organism must have been introduced into Kenya with pedigree breeding stock from South Africa and Europe. It is, therefore, to be expected that the organism will conform to the accepted characters of *B. gallinarum*.

A number of strains of the organism, selected at random from those isolated from natural cases of fowl typhoid sent to Kabete, were examined for their action on carbohydrates. The tubes used were all of the same batch and consisted of a 1 per cent. solution of the carbohydrate in peptone water with Andrade's indicator. Except for filtered dextrin they were sterilised by steaming for one hour in a Koch's steamer on three successive days. Filtered dextrin was sterilised by filtration through a Berkefeld candle. Table III gives a summary of the biochemical characters of these strains, and compares them with some other organisms, pathogenic and non-pathogenic, of the colityphoid group isolated in Kenya.

It will be seen from the table that, in marked contrast to the constant reactions of the Kenya calf paratyphoid organism first described by Daubney (1927), strains of *B. gallinarum* are rather variable in their characters, a fact on which many authors have already commented. The variations of *B. gallinarum* are, however, gradual, and do not justify separation into species. Bergey (1923) lists four species of *Eberthella* : *E. sanguinaria* (Moore), *E. jeffersonii* (Hadley), *E. pfaffi* (Hadley) and *E. rettgeri* (Hadley), the first of which is stated to be associated with cholera of chickens and the other three found in fowl typhoid. In the latest (1930) edition, *E. sanguinaria* is transferred to *Salmonella* as *S. gallinarum* (Klein), and the others are placed in Castellani and Chalmer's genus *Shigella*. The name *gallinarum* (Klein) takes precedence over the name *sanguinaria* (Moore) ; but the reason for transferring the organism to the aerogenic genus *Salmonella* is rather obscure. Hadley's paper is unfortunately not available ; but the only difference between *Shigella jeffersonii* and *Salmonella gallinarum* that can be gathered from Bergey is that the former produces acid in the unreliable carbohydrate dextrin and the latter does not. Table III shows quite clearly that the

method of sterilisation of dextrin and the actual sample in use have an important bearing on the degree of acidity produced. Table IV gives the results of testing three strains of *B. gallinarum* on the three different bottles of dextrin, all of which were obtained from one firm of wholesale chemists. The media were all prepared in the same way at the same time, and the results were read after forty-eight hours' incubation. It is obvious that without careful control dextrin is of no value in separating *B. gallinarum* from another organism.

Strains related to *Shigella pfaffi* and *Shigella rettgeri*, which ferment salicin and fail to ferment dulcite, have not been encountered in Kenya.

TABLE IV.

Dextrin	*B. gallinarum* Strain 5	*B. gallinarum* Strain 14	*B. gallinarum* Strain 24	*B. pullorum* Strain 28
Bottle A	—	—	—	—
Bottle B	—	Trace	Trace	—
Bottle C	A	A	A	—

Neither a few passages in birds nor long-continued cultivation appear to influence the biochemical characters of strains of *B. gallinarum*. Strain 5, after five passages in adult birds and three in young chicks during twelve months, showed no appreciable difference. Strain 24, which has now been cultivated since 1917, has developed no unusual characteristics. Table III indicates a danger that may arise in using modern techniques, such as inoculation into brilliant green broth followed by plating on McConkey's medium, for the isolation of non-lactose fermenting organisms from fæces and decomposed organs. Unless the organisms can be recognised on their biological characters, their pathogenicity must be tested before they can be held responsible for sickness or death.

B. gallinarum remains viable on agar slants for long periods when kept in a cool place, even when the tubes are plugged with wool and not sealed. Thus good subcultures were obtained after twenty-four hours' incubation from eleven cultures, from eight to eight and a-half months old, in which the medium had dried and shrunk almost to a stain on the tube. *B. gallinarum* has not been tested for survival in the incubator at 37° C., but it may be mentioned that the writer failed to recover *B. pullorum*, either from agar slopes or tubes of carbohydrates (some of which were not dry) that had been left in the incubator for two and a half months.

Continual cultivation of *B. gallinarum* leads to a fall in virulence. Thus strain 24, which has now been cultivated in the laboratory since December 5th, 1917, has for many years been completely non-pathogenic on intra-muscular inoculation, and is used as a vaccine. No detailed experiments have been made by the writer, but from inoculations made with a number of subcultures and

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recently-isolated cultures obtained by passage from the same strain, it would seem that subculturing favours loss or virulence. Original cultures on agar slopes do not decrease appreciably in virulence when stored for several months.

Immunity and the Agglutination Test.

Following a natural outbreak of fowl typhoid, the majority of the stock involved that recover do so completely, cease to harbour the organism, and can be shown to have gained strong immunity. In such birds the sera may agglutinate to water clear at 1 : 100 for about one month. After two or three months have elapsed the titre usually falls rapidly, so that there is no appreciable agglutination at 1 : 25. This fall in titre is not, however, accompanied by loss of immunity. There appears to be no correlation between agglutinating titre and degree of immunity. Table V, which is abstracted from an experiment on vaccination, illustrates this point.

TABLE V.

Bird	Agglutination Test				Immunity Test		Result	Cultural Examination
	Date	1/25	1/50	1/100	Date	Method		
1036	5.2.32	+ +	+ +	+	22.2.32	Fed virulent culture	died after 13 days	+ fowl typhoid
1069	5.2.32	+ +	+ +	+	22.2.32	Fed virulent culture	died after 4 days	+ fowl typhoid
479	19.2.32	—	—	—	22.2.32	Fed virulent culture	Lived	—
56	19.2.32	—	—	—	22.2.32	Fed virulent culture	Lived	—

It is now generally recognised that a certain percentage of the survivors of an outbreak continue to harbour the organism and become carriers. It has already been shown that such birds, besides being a danger to non-immune stock, are liable to die later of fowl typhoid, and from the post-mortem examination cannot be distinguished from acute cases of the disease. Carrier birds may harbour the organism in the ovary or testes (Doyle, 1926, Beach and Davis, 1927, and others) or in the gall-bladder (Martinaglia, 1930).

In 1932 an attempt was made to control an outbreak of fowl typhoid by agglu tination tests and segregation of non-reactors. A flock of about 100 pure-bred Rhode Island Red birds, mostly pullets and adult breeding stock, had suffered severely from an outbreak lasting from the middle of December, 1931, to the end of January, 1932. The birds had been kept in overcrowded runs, and when vaccination was resorted to in the middle of January it effected only a temporary check in the mortality. On March 17th, the entire flock was moved to the labora-

tory and a test was made. On the following morning, when the result was known, the thirty-two non-reacting birds were segregated in runs that had not been used for poultry for several years. In order to be on the safe side, only birds that gave no agglutination at 1 : 25 were considered clean. On April 1st to 5th, a second test was made, and it was found that in spite of the precautions that had been taken, only seventeen of the thirty-two supposedly clean birds failed to react. The value of the 1 : 25 tube was shown by the number of birds which reacted to a high titre at the second test and which had only given partial agglutination in this tube at the first. The seventeen birds were segregated and a third test was made on April 25th. Only nine birds were clean to this test, and the experiment was stopped. It must be mentioned that although the spread of infection was not checked in the experiment, the mortality stopped. Presumably this was due to a reduction in the degree of infection to which the birds that were still susceptible were exposed.

Matthews (1926) tested natural hen sera against antigens prepared from the two organisms, and found that the titre with *B. pullorum* antigen was much higher than with *B. gallinarum*. *B. gallinarum* antigen appears to be relatively insensitive to the action of agglutinating sera, and it is to this fact that the writer would attribute his own failure in the above experiment to detect carriers by the agglutination test. In confirmation of this the writer has isolated *B. gallinarum* from the ovary of a hen which was negative to the test on the day that it was killed.

In spite of the fact that no difficulty has been experienced in the isolation of *B. pullorum* from reacting hens on a farm where *pullorum* had occurred in chicks, the writer has on several occasions failed to recover *B. gallinarum* from either ovaries or gall-bladder of birds that have reacted to a high titre for many months.

After the inoculation of live culture, either virulent or attenuated by subcultivation, agglutinins first appear in the blood between the third and the seventh day. The highest titre is reached between the tenth and the fourteenth days. In the case of birds inoculated with attenuated cultures the titre falls rapidly after this time. Immune birds whose sera give no agglutination at 1 : 25, when fed with virulent cultures, will develop a titre of 1 : 100 ten to fourteen days later ; but within two to three months the sera will again be negative at 1 : 25. Subsequent feeding with a second and with a third dose of virulent culture will again provoke a high but temporary agglutinating titre. The agglutination response to the inoculation of formalised cultures is late and always poor.

Vaccination.

Vaccination by means of attenuated cultures was first introduced in Kenya by Montgomery in 1914–1915. Since that date an emulsion of living attenuated organisms has been issued as a vaccine. The vaccine is prepared from a strain of *B. gallinarum* attenuated by subculture until it no longer produces mortality on inoculation to large numbers of susceptible birds. The actual strain used has now been so attenuated for seven or eight years. The vaccine consists of a

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suspension of an ordinary twenty-four hour agar slope in 50 c.c. of physiological saline. Live organisms can be recovered from this suspension, when kept at room temperature, up to the seventh day (Walker, 1922), but it is recommended that the vaccine be used as soon as possible after receipt. The dose is 1 c.c. inoculated subcutaneously.

Reports on the use of this vaccine have accumulated now for many years and enable its value to be estimated. A large number of owners use the vaccine as a prophylactic, inoculating their entire flock every six months or every year. Reports are unanimous that in such flocks fowl typhoid never occurs. Other owners use the vaccine only when an outbreak has started. In these flocks the value of the vaccine varies. If the vaccine is used early and the birds are kept under good hygienic conditions, vaccination may stop the outbreak. If, however, the birds are exposed to a very heavy degree of infection, vaccination will only effect a slight and temporary fall in the death rate. It is therefore recommended, when possible, that birds be moved to clean runs about a week after vaccination.

Attempts to prepare vaccines of greater potency have not been successful. Kearney (1917–18) attempted to produce a high degree of immunity by giving three doses of vaccine at ten-day intervals, the second vaccine being less attenuated than the first, and the third being a fully virulent culture. This method was used in the field for a time, but as it was laborious and appeared to give no better results it was discontinued. The writer has tried formalised virulent cultures, but what little experimental evidence is available suggests that they are not as effective as the living attenuated cultures. In some outbreaks where living vaccine has failed, the use of a formalised autogenous vaccine is reported to have stopped the mortality. It is difficult, however, to appreciate the value of this evidence, for the formalised vaccine was used late and the outbreaks may have been reaching their end. Attempts to produce curative anti-sera in horses, sheep and goats were reported by Walker in 1921. The sera were, however, found to be of very low titre and to exert no influence on the course of the disease.

Summary.

(1) Fowl typhoid is the most important disease of poultry in Kenya. (2) Fowl typhoid is quite distinct from *pullorum* disease of chicks. (3) The evidence suggests that fowl typhoid was imported into Kenya by carrier birds. (4) The pathology and bacteriology are discussed. (5) The agglutination test is of no value in the detection of birds actually suffering from fowl typhoid, in the detection of carriers, or in assessing immunity. (6) Vaccination with live attenuated organisms gives good results provided that the birds are not exposed to a very heavy infection.

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MANGANESE DEFICIENCY AS A FACTOR IN BOVINE STERILITY.

By J. F. D. TUTT, F.R.C.V.S.
Winchester.

In a paper read before the Eastern Counties Division of the N.V.M.A. in October, 1933, Mr. J. R. Barker drew attention to the possible rôle of manganese deficiency in cases of bovine sterility. The present article is to show the effects of manganese therapy on three valuable Jersey cattle which, failing to respond to other treatment, in the case of two became pregnant as a direct result of this treatment.

(1) Jersey Cow ("May Star").

Previous History.—Calved third calf to full term on August 14th, 1932, and was in œstrus and served on the following dates: 28/10/32, 28/12/32, 6/2/33, 18/5/33, 14/6/33, 15/8/33, 17/9/33, 11/10/33.

Examination made on November 25th revealed a cyst in the right ovary; left ovary normal; uterus not involuted, but no discharge from cervix on massage.

Treatment.—Cyst ruptured. Prolan, 250 units (rat). Contramine, 2 c.c. Intra-uterine injection of Rivanol and glycerine, 250 c.c. The Rivanol was repeated in five days, and 50 c.c. of Yatren vaccine injected subcutaneously.

Result.—Regular returns to service. Ovaries and uterus found to be normal. Treatment with intramuscular injections of collosol manganese* (Willows) was commenced in February, 1934, and continued at intervals of seven days, until no œstrus had been observed for six weeks since service and start of treatment. Definitely pregnant; no further returns to service.

* The dose of collosol manganese was 10 c.c. per injection.

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(2) **Jersey Cow ("Post Girl")—Aged four years.**

Previous History.—Calved last calf to full term in July, 1932. Has been in regular œstrus since, but to time of first examination on July 12th, 1933, has failed to hold to repeated services.

Examination made on July 12th, 1933, revealed uterus not completely involuted. Slight cervicitis. Had been treated for vaginitis (?).

Treatment.—Lugol 1 per cent., 250 c.c. injected into uterus on July 12th, and again on July 19th, 1933. Followed by Spuman c. acid salicylic styli inserted into external os every third day.

Result.—Regular returns to service. Re-examination in January, 1934, revealed no abnormality, and manganese* therapy was commenced and continued on the same lines as in the preceding case and with the same result.

(3) **Jersey Cow ("Pure Dream").**

Previous History.—Calved normally, September, 1932. Regular, then slightly irregular, œstrus, turning at about eighteen to twenty-three days.

Examination made on March 24th, 1933, revealed large cyst, left ovary. Endometritis chiefly in the left cornu. The external os had to be dilated to admit the uterine catheter.

Treatment.—The cyst was ruptured and Lugol, 250 c.c. (1 per cent.), injected, followed by two intramuscular injections (2 c.c.) of contramine at seven-day intervals. This cow was re-examined on July 12th, 1933, and practically no change was found. The left ovary presented a smaller cyst, which was ruptured, and 500 c.c. of 1 per cent. Lugol was injected into the uterus. This was repeated on July 19th, 1933. In August a course of acid salicylic c. Spuman styli was prescribed, followed by three courses of Parat leaves, with no success. In January, 1934, the cow was again examined, and the ovaries and uterus found to be normal. Œstrus was regular, but conception did not take place. Manganese* injections were given on the same lines as in the two preceding cases, but with no success; neither did a course of wheat-germ injections induce pregnancy.

* The dose of colliosol manganese was 10 c.c. per injection.

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